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THE HARD-OF-HEARING CHILD.*

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During the last few years the hard-of-hearing child has become one of the most formidable entities for consideration within the scope and responsibilities of the otologist. The importance and far-reaching effects of this problem have been brought about by a number of valuable circumstances, each of which might be considered an independent pedagogic or scientific factor. These are:

- a.* The advent of the Audiometer.
- b.* The granting of funds by Foundations to conduct surveys in schools for the deaf and hard-of-hearing child.
- c.* The enlistment of groups of experienced otologists to conduct special surveys.
- d.* The co-operation of national associations of otologists, teachers of the deaf, organizations for the hard-of-hearing, and other groups interested in this phase of child welfare.
- e.* The tabulation and study of audiometric tests in large numbers of school children.

Reports of the results of these investigations have awakened the larger communities to the significant fact that over three million of the twenty-five million children enrolled in the public school system

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of the U. S. A. have imperfect hearing. When over 10 per cent of the future citizenship of a vast nation shows a physical defect, a large percentage of which cannot be permanently or perfectly corrected, it becomes a momentous question in which not only the otological and medical professions are concerned, but one in the careful study of which every vital agency of the commonwealth must be interested.



3-A Audiometer.

It is quite evident from the sweeping generalization "that there are three million deafened school children in the U. S. A." that active steps must be taken to meet this issue. It is even claimed that deafness is on the increase despite the fact that otological science, genetics, social hygiene and communal welfare are making rapid progress in every direction.

The Advent of the Audiometer.

In 1926 the acoustic engineers of the Bell Telephone Laboratories, co-operating with committees of otologists, recognized the fact that

new forms of apparatus especially adapted for testing the hearing of large groups of children in the public schools, with minimum expenditure of time, would be necessary.

"The first apparatus developed, the 3-A Audiometer (see illustration), was intended to replace the watch or acoumeter for making quick tests. It consists essentially of three parts connected together electrically; namely, a simple electric generator called a buzzer, an electrical network called an attenuator, and a telephone receiver. The buzzer generates an electrical current having a large number of component frequencies which are scattered throughout the audible pitch range. The attenuator is a system of resistors arranged so that



3-A Phonograph-Audiometer.

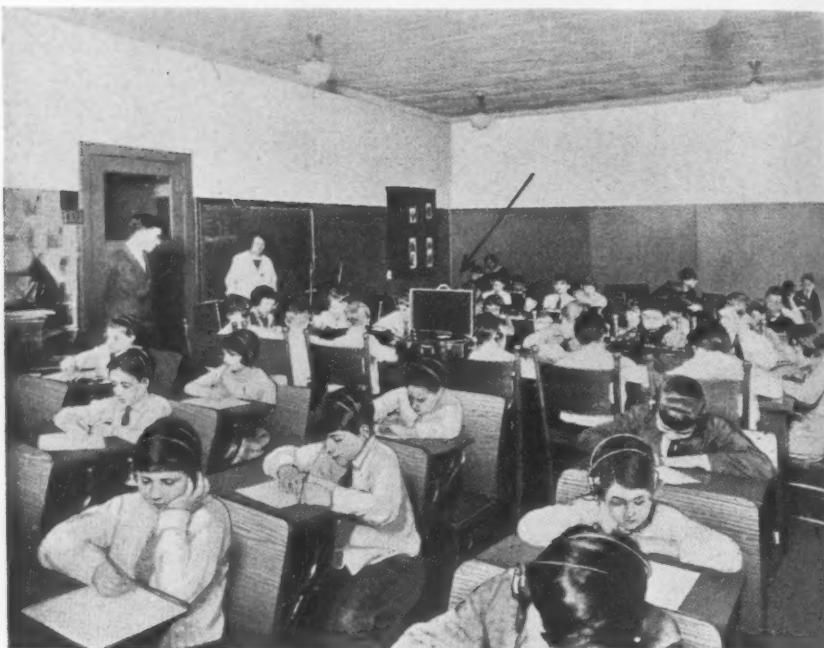
by turning a dial it is possible to change the intensity of sound issuing from the telephone receiver from the threshold of hearing to magnitudes as loud as the ear can stand. The dial is calibrated in sensation units (S. U.)¹ so that when it is adjusted to give the minimum sound that the child can hear the hearing loss can be read directly from a scale on the dial.

"Our experience with this instrument indicates that it will give results of considerably greater accuracy than can be obtained by the watch-tick test, and that it does not require specialists to operate it. After a few hours' practice anyone with ordinary intelligence can obtain reliable results."

The watch test, acoumeter, whisper and conversational voice, and other older methods for functionally testing the hearing were found

to be inaccurate, uncertain and cumbersome, and occupied too much time and personnel.

With the evolution of the *4-A Audiometer*, the product of Bell Telephone Laboratories, an apparatus was placed at the disposal of doctors, physicists, teachers and nurses, with which groups of children could be efficiently tested, the results promptly tabulated and pupils with hearing defects segregated. The entire process made possible the simultaneous testing of 40 school children in 20 minutes.



4-A Audiometer—Group Testing.

"The 4-A Phonograph-Audiometer consists of a spring-driven turntable like that of an ordinary phonograph, an electro-magnetic reproducer, and a group of telephone receivers. Light wooden trays were found to be the most convenient way of storing and distributing the telephone receivers to the children. Each tray contains eight receivers wired to jacks through which cord and plug connections are made between trays and to the reproducer."

After telephone receivers have been distributed and properly adjusted (see illustration), the children are told that they are about to hear numbers called, first by a woman and then by a man, who seem to be moving farther and farther away, so that the sounds will grow weaker and weaker. They are asked to write as many of these numbers as they can hear. The phonograph is then started and the

NAME <i>Jesse Smith</i>	AGE <i>13</i>	
GRADE <i>6 A</i>	DATE <i>Jan. 26, 1926</i>	
DO NOT MAKE ANY NOISE AS IT WILL SPOIL THE TEST		
INSTRUCTIONS		
<small>YOU WILL HEAR NUMBERS SPoken BY A PERSON WHO IS MOVING AWAY FROM YOU. THE VOICE WILL GET WEAKER AND WEAKER. LISTEN CAREFULLY AND WRITE AS MANY NUMBERS AS YOU CAN.</small>		
HEARING LOSS 1 526 368 414 111 648 526 363 538 600 131 0 -3	MASTER SHEET RECORD No. 1 TEST 1 TEST 2 526 538 525 436 624 841 482 115 856 418 416 853 626 404 363 351 528 341 634 580 124 565 058 134	
	LEFT EAR TEST 3 TEST 4 653 646 224 662 546 648 288 624 683 331 416 462 813 452 345 126 588 831 133 033 461 554 165 636	
	RIGHT EAR 525 285 868 624 841 543 482 115 225 856 418 262 416 853 444 626 404 622 463 118 445 544 238 388 344 664 642 458 8 0	
	HEARING LOSS <i>0</i>	
	<small>DID YOU EVER HAVE</small> <small>DID YOU EVER HAVE</small> <small>DOES IT HURT NOW?</small> <small>DO YOU EVER HAVE</small> <small>WHICH EAR?</small>	
	<small>EAR <i>no</i> WHICH EAR? <i>—</i> WHEN? <i>—</i></small> <small>WHICH EAR? <i>—</i> WHEN? <i>—</i></small> <small>BZZING, HISSING OR ROARING? <i>yes</i></small>	

Test Chart; Master Sheet.

first thing they hear is: "You are going to have your ears tested. Write the numbers which you hear in column 1." Then they hear numbers spoken, the loudness of each sound being less than that of the preceding one. The test is given twice by a woman's voice and then twice by a man's voice.

The receivers are then changed to the left ears and the test repeated. In the upper grades, a record of three-digit numbers is

used, while for the earlier grades the record is one or two-digit numbers.

It has been found impracticable to use this group method for testing in grades below the second half of the second grade.

Master sheets for correcting the papers are so arranged that for easy comparison they may be placed alongside the numbers written on the blank form by the child. The hearing loss for any test is found in the outside column opposite the last number heard cor-



2-A Audiometer. To make individual case test and produce Audiogram.

rectly. With the apparatus and method outlined above, it was found that 100 to 150 children, in groups of 40, can be tested per hour.

PROCEDURE IN TESTING WITH THE 2-A AUDIOMETER.

1. Preliminary testing in classes (groups of 40) in a quiet classroom, with the phonograph-audiometer and questionnaire history by a competent tester and an assistant.
2. Inspection after the regular school hours of the records made that day to determine those to be retested.

3. Retest of the borderline cases (about 18 per cent of the total), on the following day with the 2-A Audiometer and the preparation of an audiogram.
4. Careful otologic examination and diagnosis by a competent otologist.
5. The recording on a prepared sheet of all the important data from the above tests for each deafened child.
6. A note to teachers through principal advising disposal of case based on pedagogic classification.
7. A note to each parent or guardian stating that the child is slightly, moderately or markedly deafened in one or both ears, and that he should be sent to a first class ear clinic or a competent otologist to determine what can be done, if anything, to improve the hearing or prevent the progress of the disease.
8. A check-up to see whether the child has been cared for as above, and the sending of a duplicate notice to parents or guardian if it has not been done.
9. Careful filing of the records, so that the changes may be noted, and knowledge as to the detection of incipient and progress of promising cases gained.
10. Regular yearly testing of the whole school by the foregoing procedures.

The Granting of Funds by Foundations to Conduct Surveys in Work with the Deaf and Deafened.

In 1926 the Laura Spelman Rockefeller Memorial granted funds for the development of a survey to be undertaken by a committee to whom was entrusted the details of the testing of hearing of the public school children of the United States. Some of the facts gleaned by a study of the returns should be of special interest (Newhart).

Reports were tabulated from 34 cities having a population of 6,835,000 with a school enrollment of 887,000. In the school year of 1928-29, a total of 225,000 pupils were tested by means of the 4-A Audiometer. The 34 communities included 20 cities of over 100,000; five cities of 50,000 to 100,000, and nine with a population under 50,000. Geographically they were scattered from Massachusetts to California and from Minnesota to Texas.

"The most striking feature brought out by the study of the information obtained from these tests is the wide variations affecting the upper respiratory tract, by differences in the ages of the groups tested, by the handicap of language, especially among younger children of foreign parentage, by variations of intelligence and by the economic and hygienic conditions prevailing in the homes."

"We must bear in mind that the chief purpose of the tests is to single out for careful otological scrutiny those who have only a relatively slight hearing loss, in order that such children may be given the benefit of early corrective treatment when it is indicated."

Another interesting observation is that "research now being carried on in smaller communities shows a far greater incidence of hearing loss among our rural population than in the larger cities, where a program of school hygiene has long been in force.

"From what has been stated, it is evident that the use of the Audiometer in the schools, although it has been widely introduced, is still in the pioneer stage, and there remains much to be desired in the way of improving its application and in the interpretation of the results obtained.

"The outstanding conclusion which we are forced to draw from a study of the present situation is that there exists a very urgent need for standardizing the methods of using this most valuable aid in the detection of hearing loss. Not only is it necessary to standardize the technique, including the establishment of uniform bases for the retest and for notifying the parent, but there should be a serious effort made to secure uniformly effective methods of follow-up of all cases found to have hearing defects. Not until this is achieved will we be able to secure the utmost efficiency in organized efforts for deafness prevention."

Enlistment of Groups of Experienced Otologists to Conduct Special Surveys.

The *Permanent Committee on the Deaf Child*, representing all medical interests that have any bearing on these problems, created by authority of the American Medical Association and endorsed by National Otological Societies, Associations of Teachers of the Deaf, and by the American Federation of Organizations for the Hard-of-Hearing, originally consisted of Dr. Charles W. Richardson, of Washington; Dr. Thomas J. Harris, of New York; Dr. Elmer L. Kenyon, of Chicago, and Dr. Max A. Goldstein, of St. Louis.

It was through members of this committee that a plan for a survey of schools for the deaf, and later for a functional test of hearing of all public school children, was first discussed and later projected.

A committee of experienced otologists was selected to supervise these surveys, the Audiometer was introduced to the several National Otological Societies and thus the personnel and apparatus for testing were developed and the work undertaken.

Co-operation of National Associations of Otologists, Teachers of the Deaf, and Organizations for the Hard-of-Hearing.

In 1926 the American Medical Association and the several National Associations of Otologists adopted the following resolution:

"Whereas, Recognizing the fact that the most effective means for the prevention of deafness consists in the early detection of hearing impairment, thereby giving opportunity for the prompt removal of contributing causes, and, believing it to be one of the important functions of our public school authorities to safeguard the integrity of the special sense-organs, as well as the general health of the school child; be it

"Resolved, by the American Medical Association that it heartily favors the provision of our public school authorities for regular periodic examinations of the hearing acuity of all public school children, such examinations to be adequate to detect even slight degrees of hearing loss."

This resolution has been adopted by all National Organizations of Otologists, by various State and County Medical Societies, the American Federation of Organizations for the Hard-of-Hearing, the American Student Health Associations, and by other groups.

The general adoption of such a resolution may be interpreted as the forerunner of a comprehensive plan for the conservation of hearing and the prevention of deafness.

As a clinical entity, the hard-of-hearing child has been given every consideration. Tonsils have been removed *ad libitum*; adenoids have been curetted at frequent intervals; the nasal septum has been resected in many children under 10 years of age; chronic bilateral suppurative otitis media has been subjected to radical mastoid operation with resultant great loss of hearing, and frequently continued discharge from mastoid fistulae, and even with all of this ambitious surgery the proper remedy has not yet been found to reduce the percentage of defective hearing in children or to sound the keynote of deaf-prevention.

The Pedagogic Classification of the Hard-of-Hearing Child.

The pedagogic classification of the hard-of-hearing child and his educational development is of even more vital importance than his consideration as a clinical entity. Pedagogically, the classification of the hard-of-hearing child into several groups is dependent on: *a. The age of the child; b. degree of defective hearing; c. acquired fluency of speech.* In an address at the Fourth Annual Conference of the American Federation of Organizations for the Hard-of-Hear-

ing, meeting in Chicago in June, 1923, I suggested the classification of the hard-of-hearing child into several groups; further study of this classification has convinced me that my original idea is still tenable.

There are several types of deafened children with which the otologist, the educator, the social service worker and the community are concerned. One type is the congenitally deaf child who has never heard speech. This child does not enter into the present analysis, as our topic concerns only the child who has acquired a hearing defect.

There are two types of acquired deafness in children: First, the child who has acquired deafness before he has sensed fluent speech; the other, the child who has acquired deafness after fluency of speech has been established.

I. ACQUIRED TOTAL DEAFNESS BEFORE THE DEVELOPMENT OF SPEECH.

The child who has acquired deafness before speech has been developed must be offered practically the same course of training as the congenitally deaf child who has never heard speech. Arbitrarily, the child who has acquired deafness before the age of 3 years may be placed in the same class for training as the child who has never heard speech. The child of this type may be logically enrolled in a school for the deaf.

II. ACQUIRED TOTAL DEAFNESS AFTER THE DEVELOPMENT OF SPEECH.

Acquired total deafness in this group results largely from meningitis, influenza and other infectious disease in which a toxic selectivity seems to be most virulent in producing profound deafness.

For the first year or two after sustaining this serious handicap, this type of child will require intensive individual training.

It is a frequent observation that meningitis takes a greater toll in profound deafness than all other cranial nerve invasions combined. Of 100 children who suffer from the invasion of meningitis, 90 per cent are affected by deafness, and in the other 10 per cent may be included all pathologies affecting the other cranial nerves. The deafness that follows meningitis and influenza is of profound character and to those who have closely followed re-education methods, it is a significant fact that deafness resulting from meningitis is rarely susceptible to such re-education.

On the other hand, deafness resulting from measles or scarlet fever is more frequently associated with the so-called tone-islands (ton Inseln), as first described by Bezold and later followed by Urbantschitch and Goldstein, where a residuum of hearing and some functioning capacity of the acoustic labyrinth may still be retained and be susceptible to re-education. In conjunction with this observa-

tion, it is also interesting to note that a large percentage of children with biologic congenital deafness also exhibit sufficient residual hearing which may be used as a nucleus for re-education.

Every effort should be made to conserve speech in fluency, rhythm, pitch, volume and quality. Unless constantly drilled, this type of case drifts rapidly into slovenliness and inaccuracies of speech, soon acquiring the characteristic monotone so prevalent in the congenitally deaf child.

Lip-reading, of course, is also an essential of this child's training, for, with the total loss of hearing, this becomes his most precious asset for social contact.

III. DEAFNESS BEYOND THREE FEET (ONE METER) FROM SOURCE OF CONVERSATIONAL VOICE.

The source of deafness in this group is either scarlet fever, measles or other exanthemata; or, as is sometimes found, the congenitally deaf child with residual hearing; or congenital syphilis.

In this group there must always be a fixed handicap, in that the hearing distance for conversational voice may never be improved beyond three feet (one meter) from the source of sound.

In the pupil from 3 to 8 years of age the training must be individual and intensive and must be directed to a careful acquisition of lip-reading. This child, until specially segregated groups have been organized for such training, will, perhaps, find his best development possible in a modern school for the deaf.

If, after the age of 8 or 9 years, his speech is fluent and lip-reading perfect, he can be transferred to a normal schoolroom and continue his education with normal-hearing children.

IV. DEAFNESS BEYOND TWELVE FEET (FOUR METERS) FROM SOURCE OF CONVERSATIONAL VOICE.

These children should be grouped in classes not to exceed 15 of the same grade. Lip-reading and voice conservation should be given by intensive training, a special teacher assigned to lip-reading and speech, the instruction to be continued by preference in a graded school for the hard-of-hearing children. If lip-reading efficiency and fluency in speech warrants, these children may be transferred to the normal schoolroom.

V. DEAFNESS BEYOND TWENTY FEET (SEVEN METERS) FROM SOURCE OF CONVERSATIONAL VOICE.

It is advised that such children remain in the normal schoolroom and be seated in a favorable location to the speech source of the teacher and fellow pupils. Daily instruction should be given in lip-reading and the speech of the pupil conserved by special oral practice.

VI. SLIGHT IMPAIRMENT OF BOTH EARS NOT EXCEEDING A HEARING LOSS OF TWENTY S. U.

Such impairment may be caused by perforated eardrums, suppurative otitis media chronica, nasal or tubal obstruction. The hearing loss in at least one ear should not exceed 18 feet (six meters). These children should receive remedial or operative treatment if advised by the otologist and should be retained in the normal schoolroom.

Lip-Reading as a Most Vital Asset to the Rehabilitation of the Hard-of-Hearing Child.

When the cause of a serious impairment of hearing has been determined and the otologist has conscientiously advised that all forms of treatment may be of no avail toward improving the hearing, there is still one resource that remains.

It was my privilege to address an Annual Conference of the American Federation of Organizations for the Hard-of-Hearing on the significant subject, "The Deafened Child, His Handicaps and Their Correction."

For the convenience of many of the hard-of-hearing members of the conference, an energetic and enterprising ear phone company installed a system of batteries and instruments with individual headsets. During my address I noticed a flurry and restlessness in one seat section of some 30 or 40 persons, and it was found that a single ear-phone was out of commission, and the contact in the electric circuit for that whole group of ear-phone users was broken; the proceedings of the conference were interrupted until the broken connection had been re-established. Hence, my observation: A natural asset to hearing, like lip-reading, is one thing; a mechanical aid, depending on the efficiency of an electric instrument, is another.

This conference pre-eminently represented an endorsement of lip-reading and was made up largely of persons trained in this art. I took this as my text and emphasized the fact that efficiency in the art of lip-reading was a greater asset for the deafened human than the efficient performance of an electric hearing device. An ear-phone is not a constant and reliable companion; the ear-phone may get out of order; the time may come, in certain types of deafness, when an ear-phone is no longer of use; when too much dependence has been placed on the use of an ear-phone, the deafened person may find himself helpless when the ear-phone gets out of commission or when his deafness becomes sufficiently accentuated to make the ear-phone useless.

The simple and inexpensive device illustrated below serves a threefold purpose: 1. It may be used to conduct the amplified sound of voice or musical tone to both ears simultaneously; 2. the pupil,



Simplex Hearing Tube.

A and A¹, ear tips; B, aluminum funnel for speech of deafened child; C, funnel for voice of instructor; D and D¹, brass Y connectors.

by speaking through the funnel, may hear the sounds of his own voice and may be taught more readily to correct the modifications therein; 3. the funnel may be placed in contact with phonograph,

loud-speaker, organ pipe or other form of continued musical vibration, in order to stimulate the ears of the pupil by some modification of phono-massage.

Let us, therefore, emphasize the greatest asset which may be found for the hard-of-hearing individual, namely, *the efficient teaching and learning of lip-reading*.

Every human with defective hearing should make it his special objective to become a fluent and accurate lip-reader and to make this asset his most dependable one.

I plead strongly for greater efficiency in lip-reading, for the hard-of-hearing child. His task has not been accomplished when he has completed a systematic course in lip-reading with a competent teacher. He has had class practice with teachers and fellow pupils who are overly indulgent; he has been taught to watch trained lips in speech. Unfortunately, the humdrum of every-day life into which he goes does not present the same accuracies of speech and the same practiced lips to read as he has found in the classroom or with his special teacher. Too much emphasis cannot be placed on the value of conservation of speech for the deafened. In the hard-of-hearing child the voice becomes monotonous, the inflection less pronounced, the rhythm and phraseology stilted and the intensity of voice frequently much reduced because the deafened subject cannot hear the sound of his own voice and fears he is talking too loud.

It is just as important for the teacher of lip-reading to teach conservation of the characteristics of the voice as it is to teach efficiency in lip-reading.

I would not decry the use of artificial aids to hearing. There are many types of defective hearing in which a substantial mechanical aid may be of great comfort. I feel, therefore, that it is a kindly bit of advice to the deafened person not to place absolute dependence on the mechanical or electrical device selected by that person to amplify sound, for one can become so dependent upon such a device that another period of discouragement comes into one's life when that instrument or device no longer fulfills this purpose.

In the presentation of these observations I have endeavored to summarize the results of the efforts that have been made within the past decade in the U. S. A. in behalf of the hard-of-hearing child. The problem, because it involves so large a group of the future citizens of the nation, must be regarded as a very significant one and is entitled to the serious thought of every individual and group engaged in the service of child welfare.

3858 Westminster Place.

ROENTGENOLOGY AS AN AID IN THE PRACTICE OF OTOTOLOGY AND RHINOLOGY.*

DR. FREDERICK E. HASTY, Nashville.

To those who have not had the experience of studying diseases of the paranasal sinuses and mastoids by repeated Roentgenological examinations, what I am about to say may seem fanciful, perhaps somewhat theoretical, and very likely impractical. I am convinced, however, that our knowledge of disease in these structures can be very much augmented by the proper use of Roentgenology.

It is my observation that there are still many otolaryngologists who are not at all enthusiastic over the use of Roentgenology in their work. The main reason for this is that their results have not lent assistance commensurate with the trouble and expense involved.

If one has never had the opportunity of observing good Roentgenograms on all of his patients suffering from paranasal sinus and mastoid diseases, he will encounter many illuminating experiences. The otolaryngologist who does not have time or ability to study and interpret his Roentgenograms is not doing himself, his patients or his profession justice. It is fair to expect of the Roentgenologist good films and hearty co-operation in their interpretation; but it is hardly fair to expect him to give a detailed description of a film that should be desired by the man who is expected to use the information so obtained in determining the management of a particular case.

Roentgenograms poorly made and improperly interpreted will lead to many serious mistakes, and unless the work is correctly done from beginning to finish, the patient would perhaps be better off without any pretense at a Roentgenological examination. Repeated examinations, however, will frequently give valuable information as to the nature of a sinus or mastoid disease. A slight change in the position or exposure may give a much better film. Only the best Roentgenological equipment should be used in Roentgenology of the head. After the most careful work there will be some unsatisfactory results. In such instances the effort should be repeated, for one

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should not accept films that do not seem to be very close to as good as can be made.

The time is not far distant when a surgeon will be censured for operating on a paranasal sinus or mastoid without the aid of good Roentgenograms. Such a study of the diseases of the paranasal sinuses and mastoids will decrease very markedly the number of "piddling" operations and, I hope, all kinds of operations.

There are certain types of pathology of the paranasal sinuses which will produce little, if any, definite pain and likewise cause little, if any, drainage. Because of this, the patient is not likely to give a history that would lead one directly to suspect the location of pathology. This is particularly true in instances where disease about the roots of the teeth has extended to the lining membrane of an antrum.

The condition of the osseous tissues should always be considered, but in the majority of instances we are most interested in the mucous membrane lining the bony cavities of paranasal sinuses and mastoid cells. Therefore, the Roentgenograms should not be sufficiently exposed to burn out the soft tissue details. I grant that an attempt to demonstrate such details adds considerably to the difficulty of getting a film that is exactly satisfactory, for there is the greatest variation in the size of heads and the density of the bone in different individuals. This, moreover, is the main reason why Roentgenograms of children's heads have been so unsatisfactory. When a child is willing to co-operate, Roentgenograms can be obtained which are just as valuable from a diagnostic standpoint as in an adult. Of course, the difficulties encountered in obtaining these films are more because the required penetration cannot always be estimated by age or size, due to the fact that there is a very great variation in the calcium salts deposits in children of the same age and of a similar size. Then, too, the immobilization of the head is more difficult to obtain. Roentgenology should not be expected to displace clinical signs and symptoms or to predominate over the indications of a well taken and reliable history.

Roentgenology of the paranasal sinuses cannot be utilized to the best advantage without the use of some radiopaque substance. For this purpose I have found some of the iodinized oils to be the most satisfactory. I feel that the use of some such substance is indispensable, and therefore I make it a routine in irrigating a supposedly acute antrum to pump out the irrigating solution and instill lipiodol. If there is any suspicion of the sphenoid sinus, it can be very easily injected through the normal opening and examined Roentgenologi-

cally at the same time as the antrum without the shadows of either interfering with the other. Radiopaque oil injected into the antrum will give information that cannot be otherwise obtained. I am aware that many men have become discouraged with the use of radiopaque substances. This, in the main, has been due to the lack of proper technique in instilling the oil, in placing the patient in position, and in making the exposures when doing the Roentgenology. For the sphenoid I inject lipiodol and esters of olive oil, equal parts, the solution being warmed to about body temperature so as to make it thinner, and also to avoid pain that sometimes accompanies the instillation of cold fluid in the sphenoid. Then into the antrum, through or near the normal opening, I inject lipiodol and liquid alboline, equal parts, slightly warmed. The head is kept upright during both of these injections, and continuously in this position until a lateral view is made. This assures one of a true outline of the condition of the cavities. I feel that the lateral view will much more frequently be of value than the anteroposterior view. If the antra and sphenoids on both sides are to be examined, first do one side; then wash the oil out and do the other side, thus avoiding the super-imposition of the shadows of one antrum and sphenoid on the other. The chin-vertex position will give another view of both the antra and the sphenoids. If one will remember that he is dealing with a fluid and that its position will depend upon gravity, he will then have a fundamental working basis for this part of the technique.

Mastoids: With good stereoscopic films it is possible to study the mastoid cells by viewing them from above,—that is, by looking through the top of the head from below, from an external surface and from an internal surface as if looking through the head from the opposite side. Looking from above, one is able to get an idea of the general shape of the mastoid. Erosions of the internal plate may be discovered, and some outline of the lateral sinus may be made out. Pneumatization near the apex can be studied. In chronic mastoiditis one may be able to make out cavities in this position that would not be apparent in a lateral view. The same films may be turned over and studied from below. In this position the cells in the apex can be very well made out, and destruction in the bony walls of the canal of the external ear can be sought for, the jugular and other foramina in this region can be studied, and cavities in parts of the mastoid process can be better made out in this position than in the lateral view.

Lateral View: Study from the external surface will give very much the usual demonstration of the mastoid plates. The same

films turned over and studied from the opposite direction will give a good outline of the lateral sinus, will frequently demonstrate destruction in the bony wall of the lateral sinus, and may give a better view of the cavities than from the films studied externally. This method of examining the mastoid really gives one an opportunity of studying the mastoid process in four directions; and during this observation of the mastoid process, *per se*, one has also an opportunity to see the relation of other important structures near by. I think the advantage of this method of radiographing the mastoid will justify the expense and trouble.

At the very best, Roentgenology cannot be relied upon as a final indication as to whether or not surgery of the mastoid should be done. If properly used, however, invaluable assistance can be had. A great deal more information can be obtained of an acute mastoiditis if early in the infection, say at the time the ear is opened, the mastoid be radiographed and the films be filed for future reference. These early films will be of inestimable value as records for subsequent comparison as the disease in the mastoid progresses or regresses. To wait to make the first films until one feels surgery is necessary is not doing Roentgenology justice. I grant that frequently otolaryngologists do not see the patient until this state of pathology already exists, in which instance he would have to make the best of the situation. Here again one must remember that Roentgenology is not a sure way of obtaining the desired information and that repeated examinations during different stages of the pathological process would very materially enhance the value of the Roentgenological indications. In other words, just as in other laboratory aids, repeated tests are very desirable.

Ethmoid and Sphenoid Cells: To my mind, the terminology of *ethmoid* and *sphenoid cells* carries with it a more exact description of these structures than does the term *ethmoid* and *sphenoid sinuses*.

I think that to the average otolaryngologist the term *sinus* does suggest a cavity which is more or less uniform in outline and size, while the term *cell* is more likely to suggest to his mind a cavity that is irregular in outline and size; therefore, I prefer to speak of ethmoid and sphenoid *cells* rather than ethmoid and sphenoid *sinuses*. The variation in the structures in the ethmoid cells is rather commonly recognized. I think, however, there is a lack of appreciation of the variation in the structures of the sphenoid cells. Up until a comparatively recent date it has not been easily possible with Roentgenology to demonstrate the detailed anatomy of ethmoid and sphenoid cells. Now, however, with modern X-ray equipment it is

easily possible to demonstrate rather in detail the anatomy of these structures and at the same time, in many instances, to find evidence of pathological changes. It is not always easy to interpret films of ethmoid and sphenoid cells into terms of pathology. If, however, one is thoroughly acquainted with the history and clinical findings in a case, and then has good films, and has a certain amount of experience in comparing findings at operation and what was observed in X-ray films, one can learn, with reasonable certainty, to detect in the film evidence of different types of pathology. Here again one's knowledge of the history and clinical findings in a case puts him in a most favorable position. The knowledge, obtained from the films, of the anatomy of the ethmoid and sphenoid cells will within itself justify the trouble and expense involved. The study of a small series of films made in this position, I think, will convince anyone that variation rather than constancy is the rule in the anatomy of these structures, and that the variation in anatomy is frequently the pitfall for the operator who is not thoroughly acquainted with the structures in a particular case under observation.

J. C.: This film will demonstrate how in a 9-year-old child one ethmoid may be acutely and extensively involved while the other is comparatively clear. Some 10 days after the first film a re-check showed the other ethmoid also to be involved. This child was of a tuberculous family, and following an acute cold she had an irregular elevation of temperature, which caused the family and physician to fear a tuberculous infection. While the paranasal sinus infection was somewhat slow in yielding to treatment, the temperature cleared up after the infection in the sinuses decreased. Certain diseased conditions of the ethmoids can be very reliably demonstrated by Roentgenology.

C. L. G.: The first film in this case will show a cloudy antrum, but irrigation done at this time failed to demonstrate pus. The solution was pumped out, lipiodol was injected, and the second film demonstrated a filling defect. This is a typical case of what is usually termed an acute infection of an antrum. He had no pus. The severe pain must have been due to the pressure produced in the forming of a polyp, cyst or submucous abscess.

H. D. H.: This will demonstrate what may be found in an acute infection of a frontal sinus. This patient had suffered terrific pain. His temperature had been from 100° to 104° for several days. It is sometimes surprising how much involvement of the bone there may be in an acute infection of the frontal sinus, and for this reason I

have found it advisable to do repeated Roentgenological examinations of frontal sinus conditions that do not improve during treatment.

R. M. H.: This is a case of pansinusitis. Extensive intranasal surgery was done on both sides and cotton pledges were saturated with lipiodol and placed in the ethmoid region. It seemed as though we had practically destroyed the ethmoid cells. I find this to be a convenient method of marking the extent of operation in ethmoid and sphenoid surgery.

J. J. J.: This film outlines a filling defect in the anterior portion of the antrum. In studying tumors in this location it is well to bend the head slightly forward so that the lipiodol will surely come in contact with the tumor mass.

D. M. C.: This is the type of case which has been overlooked all too frequently. The teeth, which were badly diseased, had been removed; but the patient continued to show evidence of focal infection. Since, however, on transillumination the antra were clear and there was not a great deal of drainage and irrigation had been negative, competent men had pronounced the sinuses as not being the source of infection. I, too, found the antra to be negative on irrigation. These films, however, show the filling defects which were confirmed at operation. The patient showed prompt and satisfactory improvement. Too much stress cannot be laid upon the fact that lack of secretion in the nose and lack of findings in irrigation are not to be taken as positive evidence that there is not disease in an antrum. The secret of this type of pathology lies in the fact that the disease is beneath the lining membrane, and that there may be little or no accumulation of mucus and pus within the free cavity. This is frequently true in cases in which the origin of the disease is due to pathology about the teeth. The otolaryngologist and dentist must co-operate, and most carefully study their cases.

SUMMARY.

1. Roentgenology can be so used as to lend great assistance to the otolaryngologist in the study of the anatomy of many of the structures with which he has to deal.
2. While not infallible, Roentgenology is an indispensable aid in the study of the diseases in the bony cavities of the structures about the head.
3. It is the duty of the otolaryngologist to interpret Roentgenograms of his patients.
4. Radiopaque solutions are indispensable in the radiographic study of the paranasal sinuses.

5. In many instances disease in the paranasal sinuses is located beneath the lining membrane. Therefore, there is but little, if any, pus formation in the cavity.

6. Good Roentgenograms of the ethmoid and sphenoid cells will outline the anatomy, and in many instances will give valuable information of the pathological changes in these structures.

7. Roentgenology is indispensable in the practice of otolaryngology, but it should not be expected to prevail over the indications of a good examination and a carefully taken and dependable history.

1205 Medical Arts Building.

THE HARD-OF-HEARING SCHOOL CHILD.*

DR. ROBERT M. TAYLOR, East Haven, Conn.

Introduction: Examinations testing the hearing of public school children have been made in recent years by the use of the audiometer in the majority of our larger cities and towns. In these communities where the audiometer has been used it is very apparent that under the previous method of examination many children who were hard-of-hearing were passed as normal. It is now very evident that larger numbers of children, both in the elementary and high schools, are hard-of-hearing to an extent which handicaps their progress. They are not deaf in a sense that they need instruction in a school for the deaf, but they do need instruction in lip-reading or, more properly, speech-reading. The hard-of-hearing school child, who has had training in speech-reading, finds himself on an equal footing with the normal child and his school progress is not affected by his handicap.

Incidence: The 4-A phonograph audiometer was used in New York City to test 4,112 school children. The figure 9 SU was taken

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as a basis of a classification of those more deafened than normal variations would account for. In terms of distance for hearing speech, this means that those who cannot hear until the speaker comes nearer than two-fifths of the average distance are considered to have abnormal hearing. On this basis, out of the 4,112 children tested, 14.4 per cent would be classed as deficient in hearing; 3.2 per cent having defects in both ears; 11.2 per cent in ear alone. Contrary to popular belief, the child having one ear defective in hearing was found to be handicapped and likely to be retarded. Estimating the school population of the United States at 24,000,000, there are over 3,000,000 school children with some hearing defect. Of this number there are 340,000 impaired to such an extent as to need lip-reading badly (Fletcher and Fowler).

Causes of Deafness: The United States Census Bureau at Washington in 1920 made an analysis of 35,026 cases of deafness. The following causes are listed and the percentage of their incidence. The list is lacking in accurate diagnosis but gives one an idea as to causes. Congenital, 45; scarlet fever, 8.5; meningitis, 8; general sickness, 2; brain fever, 1.5; falls and blows, 1.5; typhoid, 0.5; whooping cough, 0.6; diseases of the middle ear, 11; diphtheria, 3; pneumonia, 1; catarrh, 1.5; colds, 2.

The Problem: Three Million Hard-of-Hearing School Children: A tremendous task and a staggering problem far greater than most had supposed. What are we going to do about it? The hard-of-hearing child presents a different problem from the totally deaf child. We term them hard-of-hearing or deafened, and mean those children who have learned their speech and in most cases have begun their education before their hearing was impaired. The large majority have just enough impairment so that with a little help or some speech-reading they can follow along with their classes. Many of them are considered stupid, rather than deafened. Their teachers are happily surprised to discover that with proper aid they can progress with their fellow pupils.

Indications for Speech-Reading Instruction: A 9 to 12 per cent audiometric loss calls only for special attention by the teacher. These need no lip-reading, but do need a quiet classroom and a seat up front. A 12 to 15 per cent hearing loss calls for regular lip-reading classwork perhaps two or three times a week, superimposed upon the regular classwork. The children can be taken from the less important lessons, brought together with the lip-reading teacher and then returned to their regular class work. One teacher can go from school to school and handle a large number of pupils. Lip-reading

help like this for varying periods up to three years has sufficed to let the child continue almost as a normal child. A 15 to 20 per cent hearing loss needs special training. Most of these children have good speech. They are, however, too deaf to keep up with their class and have to be taught in smaller groups by teachers who understand the education of the hard-of-hearing. They will be able to take some classes with the normal children and will play and associate with normal children and will live at home. Lastly, we have those with over 25 per cent hearing loss. These belong in segregated groups with especially trained teachers. These are the borderline cases. Many of them are now in schools for the deaf. They have speech but tend to errors of enunciation. They need the supervision of an expert in lip-reading and speech correction, but are a far easier problem than your totally deaf child.

Poor Otologic Treatment and Its Results: Deafness in school children is definitely related to the kind of medical care they get in the first 10 years of life. In communities where the economic conditions are such that parents have to think seriously before they assume the expenses of a doctor, where children get little or no medical care except for measles and scarlet fever, and sometimes not for these, we find as high as 25 per cent of deafness. In schools where the economic conditions are a little better, where the families are about equally divided between foreign and American-born, we find 15 per cent deafness. In communities where they never have to worry about the cost of adequate medical care, we find but 2 per cent of deafness. This state of affairs obviously points to better care for children in the early years of life. Adult deafness in 75 per cent of the cases is the direct result of inadequate medical care during the first 10 years of life.

I would now like to discuss the part the hospital can play in the prevention of deafness. Now hospital out-patient departments are not the most efficient organizations in the world; in fact, as a class they are very poor examples of what competent medical care should be. First, the men doing this work are not paid for their services. They should be, and then someone should be on the job to see that they do their work well. The average out-patient doctor is doing this work and holding the place on the out-patient staff simply for the purpose of keeping in line for a regular staff appointment, or else to learn the work himself. The cases drag along and drag along and finally the patient gets tired for lack of results and goes to another hospital. Now this has a very definite relation to the problem of the hard-of-hearing school child. Many children who attend

these out-patient departments are put on the chronic discharging list simply because the work is not well done when first they are seen.

Extent of Help Available: Special lip-reading classes are now operating in 44 city school systems and 60 cities maintain evening classes in lip-reading. The establishment of these lip-reading classes has not been widespread and is only a partial step in the right direction. Much credit is due Dr. Franklin Bock, of Rochester, N. Y., for the establishment of a pioneer ear clinic for school children. Here children with 9 to 12 per cent hearing loss are given front seats in the classroom, those with more than 12 per cent are assigned to lip-reading classes part time, and those with more than 25 per cent receive intensive lip-reading and speech correction. All these children should be kept in an environment of hearing children and not in a school for the deaf, where they are apt to acquire the voice and enunciation of the wholly deaf child.

Summary: Early discovery of the auditory defects is necessary if maximum benefit is to be gained from adequate medical care. The audiometer should be available for the rapid measurement of the hearing defect. The results of school surveys made with these instruments are encouraging. The hard-of-hearing school child in the great majority of cases can receive his education in the regular public school, provided certain special attention is given him by his regular teachers and that special classes in lip-reading and speech are maintained as before mentioned.

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THE BIOLOGICAL FACTOR IN ANALYSIS OF OTOSCLEROSIS OR OTITIC SENILITY.*

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It is notorious in scientific circles that exponents of various theories frequently destroy the confidence of their contemporaries by insisting on explaining everything under all conditions by their own pet theories. This can hardly be said to be truly scientific — for science, to be worthy the name, must concern itself primarily with the systematizing of acknowledged laws and truths, as verified by induction, experiment and observation.

The only justifiable attitude in such matters, I believe, is to propose a solution which seems plausible, but not to maintain that it will serve to account for all the phenomena observed in any given case.

As concerns otosclerosis, or as we might better term it, "otitic senility," we have considered many things of which we know very little, and the further we delve, the more we realize how little we know. For one thing, we have to deal with conditions of matter in the interior of an organ which we can neither observe directly, nor reproduce in our laboratories. Many of the changes taking place in the ear during the progress of otosclerosis, or otitic senility, can be approached only by extrapolation and theoretical reasoning.

Many other sciences must be considered in any accurate analysis of otitic senility. The otologist, burdened as he is with many other problems directly pertinent to his own field of research, cannot be a specialist in all sciences, and thus needs the assistance and co-operation of other talent more conversant with those distinct fields of science which are, nevertheless, essential to progress in otological research. This we see, is another reason why otologists must approach this subject with open and entirely unprejudiced minds. This applies equally, of course, to those other specialists who are perhaps less conversant with otology.

One of the specialties which has received relatively little attention in the study of otitic senility is biochemistry. Unquestionably, there are many problems in the study of otitic senility which require consideration from a biochemical standpoint. For example, the chemis-

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try of bone formation and its ultimate association with the problems of phosphorus and calcium metabolism and of hydrogen ion concentration should be studied in detail in connection with new bone formation in the labyrinthine capsule.

Preliminary to further discussion, I shall here state briefly the conclusions I have evolved from an intimate and somewhat extended study of otitic senility. I have endeavored to approach my analysis of the problems attendant to this disease with every precaution available and with as close adherence to a truly scientific approach as possible. I offer this theory, not as a master key, but as an individual contribution to our knowledge of the matter before us.

I shall first state my conclusion, and shall lay before you the steps by which I arrive at my theory. I contend that the so-called hereditary tendency of otosclerosis results from a deficiency in formation of the temporal bone in the fetus and during the early months of infancy. This causes sensitization of cartilage cells of such nature, that in adult life they become responsive to changes in the hydrogen ion concentration of the blood and of blood calcium, such as occurs temporarily during endocrine deficiency, as during lactation, or as occurs permanently, such as following oophorectomy and menopause. As the diet of the mother during pregnancy and lactation is the contributing factor in this deficiency, I believe an intensive study of the intake of calcium and of Vitamins A and D of the mother at such a time will give some comprehension of the hereditary factor and consequently give a starting point for the prevention of certain bone pathology, which I hold is a very probable source-case of otosclerosis or otitic senility.

Let us approach this subject by first considering the conditions responsible for the symptoms observed in otitic senility, then review the chemistry of the formation of similar conditions under parallel circumstances in the ear and other parts of the body.

I believe it is generally conceded that the prominent symptom of otosclerosis, or otitic senility, namely, progressive deafness, is caused directly by the intermittent although infinitesimal deposition of mineral salts about the footplate of the stapes and the margin of the oval window, eventually resulting in interference of normal vibration of the stapes in the fenestra ovalis, through which we receive the transmission of sound. This might be easily likened to the almost imperceptible deposition of calcium by trickles of lime water in earth caverns, which after long periods result in stalactites or stalagmites.

To begin at the bottom of the problem, we must first note the conditions under which mineral salts are deposited in the body. It

has been observed that the mineral constituents found deposited in otitic senility are composed of calcium carbonate and tricalcium phosphate. These are the two principal mineral constituents of normal bone, so it appears that a study of the chemistry of deposition of these compounds will lead us toward the process resulting in bony ankylosis of the stapes in the oval window.

Calcification may be visualized as follows: Serum contains calcium and organic phosphorus in solution in much higher concentration than would be possible in water on account of the high carbon dioxid tension and the colloids contained in the serum. This is true in intercellular fluids where the amount of carbon dioxid is high on account of cellular activity. It is found that when the product of the concentration of calcium and phosphorus reaches a certain point, a decrease of the carbon dioxid tension, which increases the alkalinity, brings about the precipitation of the tricalcium phosphate. When the carbon dioxid tension is low because the tissue is inactive or dead, conditions are favorable for precipitation.

The possibility of the presence of a calcium-binding substance within an area has always seemed a most attractive theory to otologists and has received much attention by investigators. Of the special substances having a high affinity for calcium that might be present in such areas as we are considering, cartilage receives first consideration. It seems probable that there exists in cartilage, a specific absorption affinity for calcium salts. It is of significance that the substances in which calcium is deposited are, in most instances, of similar physical character, being homogenous or hyaline, although of the most varied chemical composition. Hofmeister¹ advanced the hypothesis that when the cartilage or other matrix becomes saturated with calcium salts and phosphates, any decrease in the carbon dioxid tension of the solution will lead to a precipitation of calcium as carbonate and phosphate in the same proportions as found in bones and calcific deposits generally. Wells found that certain substances, when introduced into the tissue fluids, have far greater powers of absorbing calcium salts and retaining them than have others. Thus, while particles of fat, spleen, thymus, etc., are found to contain, after a stay of fourteen weeks in the peritoneum, only about 12 m.gm. of calcium; a similar piece of cartilage has absorbed 154 m.gm. and this is true even if the cartilage be boiled so there can be no question of vital activity².

In this connection, it is well to call attention to the anatomy of the tissues here considered. The temporal bone is a composite bone, the petrous and mastoid parts and the styloid process being of intra-

cartilaginous origin, while the temporal squama and the tympanic part are of intramembranous origin. During the eighth week of fetal life, a center of ossification appears in the membrane in the temporal region, and the bone formed from this center subsequently unites with the petrous part and becomes the temporal squama. Another center appears in the membrane to the outer side of the periosteal capsule and produces a ring of bone around the external auditory meatus, which fuses with the petrous parts and form the tympanic parts of the adult bone². In these tissues there are to be found normally many islands of cartilage which are found to have become ossified in otosclerosis or otitic senility. Recently, Bast⁴ has given us a paper on the embryology of the capsule, while Mayer⁵ has described in detail the histology of the temporal bone in otitic senility. So our next problem is to observe some of the circumstances which alter calcium metabolism and cause pathologic deposition of calcium in the body.

The relation of the endocrin glands to calcium metabolism and its possible connection with the bony ankylosis of the stapes in otosclerosis has been previously considered by Warwick and Stevenson⁶. With regard to this influence of pregnancy, there is a feeling that this has been exaggerated. In 835 cases of otosclerosis observed by Nager⁷, nearly one-half were unmarried. Furthermore, the sex ratio was observed to be one male to 1.8 females. These observations tend to counteract the tendency to the former belief that pregnancy and ovarian disorders were invariably concurrent with otosclerosis. This does not deny the possibility of pregnancy affecting an otosclerosis already existing, for it was observed by Nager that in 46 per cent of his cases, a change for the worse was noted during pregnancy.

Fraser⁸ says that there is too great a tendency to attribute otosclerosis to one cause alone, a view to which I subscribe. I maintain that otitic senility results from a disturbance of calcium metabolism, but that the calcium metabolism deficit may result from different causes. "Among the various diseases resulting in bone destruction and redeposition," according to Fraser, "arthritis deformans merits consideration. I hold that an attack of otitis media is comparable to the match that lights the fire, and a familial cellular idiosyncrasy of the temporal bone and the female sex corresponds to the inflammable material."

It is maintained by good authority⁹ that there are many infections in the course of which recognizable organisms lodge in the tissues of the joints and produce acute or chronic forms of arthritis. This

is particularly true of the pyogenic micrococci, such as are found in suppurative otitis media. Thus, there are infectious forms of arthritis usually recognized to be the sequelae of the existence of a focus of infection elsewhere. It was noted¹⁰ that in such forms of arthritic adhesions, complete ankylosis of bony character may often occur. This is the form termed "arthritis deformans," in which hypertrophic or degenerative changes in cartilage and bone are accompanied by extraordinary new bone formation in such a way that the bone forming the joint is profoundly affected. Mayer¹¹, in his detailed description of the pathology of otosclerosis, noted areas of sclerosis throughout the labyrinthine capsule, although predominating at the margin of the fenestra ovalis; he also mentioned that in some cases the fenestra rotunda was completely ossified. He maintains, as we did, that the old bone is absorbed by osteoclasts and a new bone marrow formed and new bone is laid down in this marrow. He states that this new-appearing bone is never seen in the normal labyrinthine capsule, or in any other pathological conditions, and that it is evidently characteristic of otitic senility. As for the histology of arthritis deformans, the bone shows, for a time at least, no morphological evidence of rarefaction, but the bone marrow becomes converted into an edematous fibrous tissue with many osteoblasts and capillaries. This invades the cartilage present, causing its ossification as it goes. The activity of the osteoblasts causes much new bone formation within, and overspreading the cartilage.

It has been believed, with a considerable amount of probability, that a disturbance of metabolism will produce or aggravate otitic senility. The effects of the glands of internal secretion have been considered in this connection⁶. A bone disease which is greatly influenced by calcium and phosphorus metabolism, and which is independent of any disturbance of endocrine functions, is rickets. It is well established that rickets result from a deficiency of a food accessory, and which is usually attributed to the fat-soluble Vitamins A and D. The mixture formerly called Vitamin A has been separated into two active constituents, called A and D, each of which have similar properties in causing an increase in calcium metabolism.

My observation that a number of my patients with otitic senility gave a history of having been weaned from the breast and fed on sweetened condensed milk at a very early age, led me to believe deficiency of Vitamins A and D are contributing factors to the initial lesion in this disease.

Consideration of the selected data presented below seems to prove that a deficiency in the fat-soluble Vitamins A and D, such as may

result on changing from mother's breast milk to cow's milk, and particularly to sweetened condensed milk, will readily induce bone pathology, such as osteoporosis and rickets.

The most prominent difference in the metabolism of fresh and condensed milk appears to be the relative absence in condensed milk of the fat-soluble Vitamins A and D, which are very important factors in calcium metabolism. These two vitamins are found most abundantly in cod liver oil, butter fat, egg yolk, liver, leafy vegetables and yellow-pigmented vegetables generally. These vitamins are readily oxidized at high temperatures, but are relatively stable under the conditions generally maintained in cooking foods. A great deal of research has been carried out in connection with milk metabolism in infants and its relation to bone formation. Washburn and Jones¹² noted that sweetened condensed milk, though showing an abundant nutritive efficiency as measured in energy units, proved an undesirable food for the growing young. When the sweetened ration was used, the bones were but two-thirds as strong as were those produced on normal milk. Chi Che Wang, Witt and Fletcher¹³ observed that a change from breast to cow milk was invariably accompanied by an increase in the quantity of calcium excreted, and that the percentage utilization and retention is greater for breast milk; it is maintained by Jozef Dubiski¹⁴ that sweetened condensed milk is not a complete nutrient for the growing organism on account of the deficiency in vitamins. Sekine¹⁵, on noting the nutritive defects of canned milk, states that the vitamins are destroyed in the process of evaporating and canning, making condensed milk undesirable as infant food after weaning. This conclusion is substantiated by the experiments of Variot¹⁶. The observations of Kovenchevskii and Carr¹⁷ in experimenting with rats, were that they grew and developed normally and had a normal skeleton on a diet of cow's milk *ad lib.*, with the addition of the requisite amount of calories in the form of carbohydrates. Though the calcium content of the skeleton was normal, histologically there was osteoporosis. The experiments of McCollum, *et al.*,¹⁸ showed that a diet deficient in calcium and in fat-soluble Vitamins A and D fed the rats from birth resulted in the tests growing poorly, or not at all, their teeth became loose, the incisors broke readily, the eyes were normal, and in the bones the histologic structure was distinctly that of osteoporosis and rickets. Borst¹⁹ fed rats on a diet of skimmed milk, and consequently deficient in the fat-soluble vitamins. He noted extensive osteoporosis and atrophy of the bones, atrophy of the thyroid, hypophysis and thymus, and marked pathologic changes in ovary and testicle. It was found by Outhouse, Macy, Brekke and Graham²⁰ that fresh

raw cow milk may contain adequate amounts of Vitamins A and D to produce satisfactory growth in the rat, but this small quantity does not always protect against secondary pathologic conditions: at autopsy either single or double mastoid involvement was found in 77 per cent of the rats which had received a diet of raw milk.

In view of these facts just stated, a discussion of the vitamin reserve of the newborn will be of advantage here. The vitamin requirements of the young are exceptionally great. The newborn are fortified with a large vitamin reserve, which has been supplied by the mother. The colostrum and milk intake help to maintain a vitamin reserve during the first few months of life, but mothers that are nourished on vitamin-deficient diets are apt to have improperly developed babies. Such cases were reported by Brown, Courtney, Tisdale and McLachlan²¹. Two cases of rickets were observed as occurring in breast-fed infants and found to result because the mother's diet during lactation was lacking in vitamins and certain inorganic constituents required for normal bone development in the child. The effect of the mother's diet as a safety factor for infant nutrition is substantiated by McCollum and Simmons²². It was observed by Kovenchevskii and Carr²³ that when the mother's normal diet during pregnancy was enriched with foods high in Vitamins A and D and an excess of calcium, there was a marked decrease in the disorders of general nutrition and in the rachitic changes in the skeleton produced in the young by a diet deficient in the fat-soluble factors.

This review of the effect of dietary deficiencies on the metabolism of calcium in infants led me to further observations that a number of my otosclerotic patients give a history of having been put on a vitamin-deficient diet at a very early age, particularly onto sweetened condensed milk, which is low in Vitamins A and D. In connection with osteoporosis having been observed histologically in the bones of infants on a diet deficient in accessory factors regulating calcium metabolism, I wish to suggest that this osteoporosis and bone cell sensitivity may have been present in the temporal bone and stapes since a very early age. This osteoporosis of otosclerosis resembles but is not identical with rickets. Such an osteoporosis is more readily found in offspring when the diet of the mother during lactation has been deficient in calcium and in Vitamins A and D. As we have much evidence that storage of these vitamins in the body is possible and that it depends largely upon the food consumed, we must expect that the content of fat-soluble vitamins in a given organ or tissue may vary considerably with different individuals. This means not

only variability among the different specimens of a different type of food, but also the further complication of variable stores of the vitamins in the body of the persons according to their age, previous feeding, and possibly other conditions. In the experience of Kovenchey-skii and Carr²³, much smaller amounts of the vitamin-containing foods are required when such foods are supplied from the start as a preventative than when given as a supplement after severe nutritive decline has set in.

As the mother is responsible for the food factors contributing to the growth of the child before and after birth, it would seem that regulation of her diet, particularly during pregnancy and lactation, would influence the deposition of calcium and the sensitivity of bone cells in the child. In general, it is conceded that the richest food sources of the Vitamins A and D are, first, milk in all forms, eggs, butter and green vegetables, followed by some roots, the embryos of seeds and the glandular organs of animals, then by roots and seeds generally, and muscle tissue meats and, finally, by artificially refined products, such as white rice, patent flour, sugar and many of the refined fats which are particularly devoid of these vitamins. McCollum has repeatedly emphasized the fact that diets composed essentially of breadstuffs, meats, sweets, tubers and many fats are likely to contain too little of these vitamins and he has classified milk and green vegetables as protective foods because of their richness in Vitamins A and D, as well as in calcium.

As we have no evidence of synthesis of these vitamins in the animal body, it is probable that whatever we obtain through animal products has originally come from plants. It is from her diet that a woman receives her supply of vitamins and mineral for the increased demand in pregnancy and lactation. Thus it appears that if a child is not supplied at birth with a reserve of vitamins and minerals for bone-building, and is not supplied with it from the mother's milk, the deposition of bone will be faulty.

Now, from the data herein presented, allow me in conclusion to restate the hypothesis which I laid down in the beginning, and in support of which I offer the foregoing evidence.

It is my conclusion that the so-called hereditary tendency of otosclerosis results from a deficiency in formation of the temporal bone in the fetus and during the early months in infancy. This causes sensitization of cartilage cells of such nature in adult life that they become responsive to changes in the hydrogen ion concentration of the blood and of blood calcium, such as occurs temporarily during endocrine deficiency, as during lactation, or as occurs perma-

nently, such as following oophorectomy and menopause. As the diet of the mother during pregnancy and lactation is the contributing factor in this deficiency, I believe an intensive study of the intake of calcium and of Vitamins A and D of the mother at such time will give some comprehension of the hereditary factor and consequently give a starting point for the prevention of certain bone pathology which I hold is a very probable source-case of otosclerosis or otitic senility.

REFERENCES.

1. HOFMEISTER: Arch. Exp. Path. and Phar., 28, 210, 1891.
2. MACCALLUM: Textbook of Pathology (Saunders), 110-111.
3. BAILEY: Textbook of Embryology.
4. BAST: THE LARYNGOSCOPE, 38, 665, Oct., 1928.
5. MAYER: Wein, Klin. Woch., 37, 131, Feb. 7, 1924; *Jour. Laryngol. and Otol.*, 43, 843, Dec., 1928.
6. WARWICK and STEVENSON: Acta Oto-Laryngologica, ix, 3, 317-324; and Otosclerosis, 301, 401, 658, 683, 1929, Amer. Otol. Soc.
7. NAGER: *Jour. Laryngol. and Otol.*, 43, 15, Jan., 1928.
8. FRASER: THE LARYNGOSCOPE, 1923.
9. MACCALLUM: Loc. Cit.
10. MACCALLUM: Loc. Cit.
11. MAYER: *Jour. Laryngol. and Otol.*, 43, 843, Dec., 1928.
12. WASHBURN and JONES: Vermont Agr. Exp. Sta. Bull., 195, 6-134.
13. CHI CHE WANG, WITT and FLETCHER: *Amer. Jour. Dis. Children*, 27, 352-368, 1924.
14. DUBISKI: Roczniki Nauk Rolniczych, 11, 370-403, 1924; Chem. Zeit., 1, 1142-1143, 1925.
15. SEKINE: *Jour. Tokyo Chem. Soc.*, 42, 465-473, 1921.
16. VARIOT: Bull. mem. soc. med. hop., Paris 46, 838-844, 1922.
17. KOVACHEVSKII and CARR: *Biochem. Jour.*, 17, 187-293, 1923.
18. McCOLLUM, ET AL.: *Amer. Jour. Hyg.*, 2, 97-106, 1922.
19. BORST: Centr. Allgem. Path., 33, 306-327, 1923.
20. OUTHOUSE, MACY, BREKKE and GRAHAM: *Jour. Biol. Chem.*, 73, 203-208, 1927.
21. BROWN, COURTNEY, TISDALL and McLACHLAN: Arch. Ped., 39, 559-566, 1922.
22. MCCOLLUM and SIMMONS: *Amer. Jour. Physiol.*, 46, 275-313, 1918.
23. KOVACHEVSKII and CARR: *Biochem. Jour.*, 18, 13-18, 1924; 19, 112-116, 1925.

913-14-15 Medical Arts Building.

AGRANULOCYTIC ANGINA.*

DR. JAMES W. BABCOCK, New York.

Although this subject has been presented within the past year before this body, and its history and findings so ably discussed by Dr. Rosenthal, I felt that it was of sufficient interest to be brought before us again as a reminder that we must be on the alert for its ominous symptoms. I have to report three cases recently under my observation.

A summary of the case reports which I have been able to obtain to date reveals the following data: This disease is chiefly prevalent in women, occurring three times as often in women as in men, and chiefly between the ages of 50 to 60 years, although a few cases have occurred in childhood. The mortality is, roughly, 90 per cent. The cases recovering have had less toxicity and a higher count of white blood cells. The bacteriology is so varied as to seem non-specific. The pathology, with the exception of the lesions in the mouth and, occasionally, in other mucocutaneous junctions, such as the rectum and vagina, seems principally to be a change in the marrow of the long bones, it becoming red and soft and showing no production of new white blood cells.

The lesions usually found are some necrosis in the pharynx, tonsils, gums, epiglottis and, occasionally, in the vagina or rectum.

The treatment has been varied, but the only measures which seem effective are direct whole blood transfusions, 200 to 500 c.c. daily, or each two days, and the oral administration of nucleic acid, a derivative of yeast. Radiotherapy of the long bones seems of doubtful benefit. In fact, the few cases of recovery seem to be due more to the mildness of the attack than to any therapeutic measure. Some cases of apparent recovery died of fairly prompt recurrence.

The local lesions are best treated by cleanliness. Avoidance of any trauma, such as tooth extraction, seems important.

The underlying cause of the failure of the protective white blood cell production in these cases is not clear. Several have followed the use of arsenic, as in arsphenamine. Some, 37 per cent according to Kastlin, were afflicted after long ill health, while 63 per cent were attacked in the midst of good health. Benzol poisoning and over-exposure to Roentgen rays seem also to press the production of

*Read before the New York Academy of Medicine, Section of Laryngology and Rhinology, May 27, 1931.

white blood cells. Other changes in the composition of the blood may be associated with ulcerative lesions in the mouth. Acute lymphatic leukemia, aleukemic leukemia, an overwhelming sepsis with leukopenia, all may show mouth lesions. Infectious mononucleosis shows a preponderance of lymphocytes and some associated ulceration of the throat. I have the privilege of reporting three cases of this disease.

Case 1: Mrs. J. W. T., age 45 years. Seen with Dr. Geylin. Patient had just recovered from a suspected attack of typhoid fever but without *B. typhosus* being isolated. Three days before she was seen her temperature was 101° F., and a blood count taken then showed: W. B. C., 5,000; polys., 2 per cent; lymphocytes, 98 per cent. On the following day her throat was slightly sore. Her temperature was 104° F. A spot on the left tonsil was noted and a culture taken proved negative for diphtheria. At that time the blood count showed: W. B. C., 2,000; small lymphocytes, 100 per cent. When seen by me her throat lesions were said to be more advanced and she had some dyspnea. The possible need of a tracheotomy was discussed but this was not deemed desirable. On examination her left tonsil and its pillars were of a grayish color and there was edema of the uvula. The grayish color extended to the epiglottis and part of the arytenoids, with slight edema. There was slight, soft swelling of the left side of the neck. The patient died that night.

Case 2: Mrs. C. E., age 32 years. Seen with Dr. D. Elmendorf and Dr. K. McAlpin. Patient had had an exophthalmic goitre for five months. She had improved under treatment but was worse after a mild attack of tonsillitis one month previously. Removal of the tonsils and adenoids was advised and the operation was performed, without untoward incident, on Nov. 8, 1929.

On Nov. 22 she had a temperature of 105.6° F. There was a tender lymph node in the left cervical region. The lingual tonsil was swollen and red, with areas of white exudate there and on the tip of the epiglottis. Blood count the next day showed: W. B. C., 1,800; small lymphocytes, 100 per cent. After several transfusions and injections of protein, the patient died on Dec. 3. There was a suspicion of pneumonia in the left lower lobe and the last blood count taken showed: W. B. C., 925; all lymphocytes.

Case 3: Mrs. G. W., age 60 years. Patient was seen on Aug. 20, 1930. She had been complaining of a sore throat and mouth for four days. There was a slight fever at the onset but the patient had none when first seen by me. A blood count taken six weeks before the onset had shown simple anemia. Upon examination the nares

were found to be congested, there were three lower teeth with necrotic periosteum and the pharynx was red, with small, white ulcers on the posterior pillars. Smears were negative for Vincent's organisms. A blood count showed: W. B. C., 1,200; polys., 10 per cent; small lymphocytes, 86 per cent, and large lymphocytes, 4 per cent. After repeated transfusions, under the care of Dr. Lamb, Dr. Bull, Dr. Thompson and Dr. Sheldon, she showed, on Sept. 3: W. B. C., 3,600; polys., 54 per cent. Since then she has had her diseased teeth extracted, with dread on her physician's part, as such procedures have had, at times, a bad effect on the blood picture. One month ago she declared herself to be feeling better than she had in two years, and her blood count on April 3, 1931, was: W. B. C., 7,000; polys., 75 per cent; lymphocytes, 25 per cent. I had hoped to report this case as a recovery, since she was well at the time of the preparation of this article, eight months after her first attack. Dr. Sheldon, however, told me of the death of the patient on May 10, due to a recurrence of five days' duration, without preliminary illness and with no mouth lesions. She had malaise, a high temperature, 104° to 105° F., and she did not respond to transfusions.

The only object in making these reports is to remind the Section that several diseases may have their first symptoms in the throat and should be recognized. It seems as important to have a blood count taken in a suspicious sore throat as it is to have a culture taken to exclude diphtheria.

REFERENCES.

- THOMPSON, WM. P.: Nelson's Medicine.
KASTLIN, G. J.: *Amer. Jour. Med. Sci.*, 173:799, 1927.
LOVETT, B. R.: *Jour. Amer. Med. Assn.*, 83:1498, 1924.
SCHULTZ, W.: Deutsche Md. Wok., 48:1495, 1922.

20 East 53rd Street.

REPORT OF A CASE OF RETROPHARYNGEAL- ESOPHAGEAL ABSCESS.*

DR. JULIUS I. KLEPPER, New York.

This case is presented here because it demonstrates an extensive abscess starting in the nasopharynx, gradually working its way down towards the esophageal wall and down to the superior mediastinum. Literature on retropharyngeal abscesses is abundant but description of a combination of retropharyngeal and esophageal abscess as found in the present case is very rare.

We are fortunate to be able to show you the process of extension with X-rays and to describe the stormy period through which the child passed before it came to our attention, the emergency operation and his recovery.

The patient, L. M., was a child of 19 months, who became ill Dec. 26, 1930. He was under the care of Dr. K., who said he found the child sick with enlarged glands of the neck. The neck was stiff and turned to the left; the temperature was 105°; the tonsils were diseased and the pharynx and ears inflamed. After six days of treatment, the temperature and the torticollis subsided but the glandular swelling remained. The glands on the right became larger.

During the second week of the onset of the child's sickness the temperature was between 101° and 102° and the torticollis disappeared. There was no difficulty in swallowing fluids; solids were not given.

At the beginning of the third week the temperature subsided and the patient was doing so well that the physician permitted the child to be taken out for air. Towards the middle of the week the throat was re-examined and found paler and negative to pharyngeal digital examination, but there was no further change in the glandular enlargement. The following day the temperature rose to 104°. That night the mother noticed difficulty in breathing. The child refused all food, coughing with even slight amounts of liquid.

On Jan. 15, 1931, finger examination revealed an obstructive mass deep in the pharynx. The physician suspected a low retropharyngeal abscess and referred the child to a specialist, Dr. S. An examination was made under ether and an incision made in the right side but no pus was obtained.

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At the end of the third week the condition became distinctly worse. The child had great difficulty in breathing and was taken to a hospital in the Bronx. The blood count now showed: W. B. C., 31,000; polymorphonuclear, 92 per cent, and hemoglobin, 80 per cent. The next day, quoting the doctor, "The child looked pretty bad, but he still took some nourishment." His temperature was below 100°; Dr. S., who was in charge of this child, decided to wait and watch. Saturday a pediatrician, Dr. B., was called in; he considered the case very serious. An X-ray was taken on the following



day, showing a large mass extending to the upper part of the superior mediastinum and anteriorly $3\frac{1}{2}$ c.m. beyond the normal limit. At this time the blood count showed: W. B. C., 25,000; polymorphonuclear, 85 per cent, and hemoglobin, 70 per cent; the urine showed a trace of albumin, pus, casts and red blood cells.

The same day another well known laryngologist examined the child and also advised a waiting policy, but in view of the danger of asphyxia he suggested that it should be removed to a hospital better equipped for such emergencies. He recognized that the child's condition was very serious and that the prognosis was bad.

On Monday Dr. S. re-examined the child before he was taken to the Manhattan Eye, Ear, Nose and Throat Hospital. Here it was entered as a private case under our care, at about 9 p. m.

At 9:45 p. m. I saw the child for the first time and insisted upon examining it, only in the operating room, the same fully prepared for any emergency and for tracheotomy if necessary. It was evident to all of us that danger was imminent. The child was gasping for breath; anything disturbing could easily have proven fatal.



The examination of the child in the operating room showed: On palpation of the pharyngeal wall, posterior to the arytenoids, in the median line, a distinct mass going far down in the posterior wall of the esophagus. The upper part was the size of a crab apple. An immediate operation was advised. The parents were terribly distressed and after much consideration and persuasion by the family physician consented—only after they were told the child might last but a few minutes, without intervention.

The child was then placed in the Trendelenberg position with head well over the end of the table. An incision was made with sharp pointed scissors with the finger on the mass. This incision was carried below the cricopharyngeus muscle. No general anesthesia was used as we considered the same dangerous.

After the incision was made, pus came out under strong pressure. Suction was constantly applied by the assistant. The child spat out a good deal of pus and the rest, about four or five ounces, was removed by suction. The child immediately began to breathe more easily. With the finger massaging the esophagus for a few minutes more pus was expressed and the child showed improved breathing, so that a tracheotomy no longer seemed necessary.

The patient was then carried to bed. He was kept all night with head over the end of the bed and suction was used very frequently.

The next morning the patient was better but his condition was still none too good. A re-examination showed thickening, edema and marked fluctuation still present below the incision.

At this examination two additional incisions were made for more drainage, about one-half inch in size, a half inch apart, below the original incision, where the fluctuation was more prominent.

The day after the operation the nurse's chart showed much slower and easier respiration. The child slept at intervals. His condition was apparently better. Suction was used as often as necessary.

Subsequent culture from the wound showed staphylococcus albus and streptococcus viridans. The patient was able to swallow liquids and soft foods. The temperature was 100°, respiration 24 and pulse varying from 120 to 130. An X-ray taken on the third day after the operation showed a marked decrease in the size of the retropharyngeal-esophageal abscess.

Dr. Imperatori was called in four days after the operation, on Jan. 23, and thought the child was doing very well. But on Jan. 24 the temperature rose to 104°.

Dr. Mackenty was called in to see the child. He agreed that the cervical adenitis accounted for the fever and also suggested a blood transfusion, which was given the same day. The child's heart, lungs and urine were negative. Subsequently the child became hoarse and coughed. This lasted four days, the temperature varying from 101° to 104°. It is interesting to note that at this time the mother was also confined to bed for a week with a severe cold; this may be only a coincidence.

At this time finger examination of the child did not show any increase in the size of the abscess. On the fourth day after rise of

temperature the same dropped to between 99° and 100° and remained so until the day of discharge, Feb. 4, 1931. Repeated finger examinations of the child's pharynx and esophagus revealed that the posterior wall was still thicker than normal, as corroborated by the X-rays, which showed slow, gradual decrease in the thickness and was therefore favorable to the child's recovery.

A week after the operation the X-ray still showed a thickness of 2½ c.m., the normal thickness being one c.m. There was a further but slow diminution of thickness, of three to five m.m. on Jan. 30, and also Feb. 3. The next X-ray still showed a thickness twice that of the normal.

The blood examination before transfusion, Jan. 21, 1931:

	Differential Leukocyte Count:
Red corpuscles: 3,100,000	
Leukocytes: 15,800	Small lymphocytes: 15 per cent
Hemoglobin: 50 per cent	Large mononuclear: 6 " "
	Polymorphonuclear neut.: 78 " "
	Eosinophiles: 1 " "

Two days after transfusion:

	Differential Leukocyte Count:
Red corpuscles: 3,840,000	
Leukocytes: 11,000	Small lymphocytes: 30 per cent
Hemoglobin: 68 per cent	Large mononuclear: 3 " "
	Polymorphonuclear neut.: 67 " "

Jan. 26, 1931, blood report shows:

	Differential Leukocyte Count:
Red corpuscles: 3,780,000	
Character of red cell: Abnormal white cells, few myelocytes	Small lymphocytes: 65 per cent
Leukocytes:	Large mononuclear: 10 " "
Hemoglobin: 65 per cent	Polymorphonuclear neut.: 24 " "
	Eosinophiles: 1 " "

Four days after blood transfusion, Jan. 30, register the biggest gain:

	Differential Leukocyte Count:
Red corpuscles: 4,390,000	
Leukocytes: 7,500	Small lymphocytes: 61 per cent
Hemoglobin: 78 per cent	Large mononuclear: 17 " "
	Polymorphonuclear neut.: 22 " "

On Feb. 3, 1931:

	Differential Leukocyte Count:
Red corpuscles: 4,400,000	
Leukocytes: 12,000	Small lymphocytes: 52 per cent
Hemoglobin: 81 per cent (steady increase)	Large mononuclear: 14 " "
	Polymorphonuclear neut.: 34 " "

It is interesting to note that previous to the operation the urine examination by the family physician showed a glycosuria varying

from 0.5 per cent to 2 per cent. No sugar was present in any of the urine specimens examined for 10 days after the operation. However, on Feb. 2, 1931, the only abnormality found was the presence of 0.5 per cent sugar. The following day it was 0.4 per cent. But the blood sugar was normal 75 m.gm. per c.c. of plasma.

After the child left the hospital he was re-examined by Dr. B., a pediatrician, who found that the sugar in the urine was pentose; this is harmless. Dr. B. was also very much pleased to find that this child had recovered, as he considered the condition almost fatal. Since the child's removal from the hospital several X-rays were taken.

Monday, March 23, the last X-ray was taken. Dr. Low reports: "There seems to be considerable diminution in the thickness of the posterior pharyngeal wall since the previous examination."

This wall is rapidly approaching the normal. The X-ray of the sinuses reads as follows: "The frontals are absent. The ethmoids show a severe involvement from dense granulations with considerable absorption of the cell walls. Both antra show a moderately severe involvement from granulations. The sphenoids are undeveloped."

The cause of this child's retropharyngeal-esophageal abscess lies in the tonsils, adenoids and also the accessory sinuses.

24 West 85th Street.

Wessely, in the Zeit. f. H., N. u. O., 28:167:1931, makes a further contribution to the understanding of intracranial complications following tonsillitis and peritonsillitis. Autopsy material proved to him that the pathway of infection was commonly as follows: The inflammatory process spread in the anterior pharyngeal space tending to go upwards and reached its greatest intensity at the base of the skull where the musculature of the pterygoids and the less connective tissues of the parapharyngeal spaces are completely destroyed by the abscess formation. From the anterior border of the M. pterygoideus internus the pathology spread directly to the third branch of the trigeminal nerve and was also closely associated with the middle meningeal artery through the foramen spinosum in the middle fossa. It is by following this pathway that an infection in the neighborhood of the tonsils is able to reach the inside of the skull.

KELEMEN.

LARYNGEAL STENOSIS; CASE PRESENTATION.*

DR. JULIAN DE ARMAS, Philadelphia.

A child, age 4 years, seemed to have a cold one morning in October, 1929. Later the same day some difficulty in breathing developed, and a physician was called and diphtheria antitoxin was administered. At 8 p. m. the same day the child was taken to the hospital, where an emergency tracheotomy was performed.

Two months later, as all attempts at decannulation and corking were unsuccessful, he was sent home, wearing a cannula since no air would go through the larynx. In this condition was the child admitted to the Bronchoscopic Clinic, Graduate Hospital.

External examination of the patient showed: Tracheotomy tube, *in situ*, high; dyspnea on exertion; voice very hoarse; very little air passing through the larynx when the tube was corked; cough, with abundant mucopus through the tube, crust formation and blood at times.

Direct Examination: The lumen of the larynx was found to be almost obliterated. Through the small opening the tube was seen to be close to the vocal cords.

Preliminary Treatment: In this case it was necessary to perform a new tracheotomy in order to place the tube below the conus elasticus, eliminating the source of irritation.

Whenever a high tracheotomy has been done, a secondary low tracheotomy must take its place before any treatment is intended, because, as paradoxal as it may seem stated in this way, tracheotomy cures a stenosis produced by tracheotomy; and it is so for two reasons: First it places the larynx at rest; and second, enables treatment.

The fear of the supposed danger of the low tracheotomy is the cause of a high one, and it is not at all surprising to see them done through the thyroid and cricoid cartilage, really thyrotomies.

External examination and direct laryngoscopy should be carefully done, since the final result in the treatment of laryngeal stenosis depends upon the integrity of the cartilaginous framework of the larynx and trachea.

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Laryngoscopic Treatment: Direct dilatation was started immediately after the wound healed and the tracheobronchial condition subsided, and not before, because any foci of infection in the upper air passages is a distinct contraindication for the treatment.

The technique was as follows: The patient was placed in the Jackson direct laryngoscopic position, the larynx was exposed with a laryngoscope and straight metal dilators were passed through the stenosis, beginning with the smallest one that would pass, but with a slight resistance. As the opening is increased so are the sizes of the dilators.

When the laryngeal opening reaches the point where sufficient air can pass through to enable the patient to breathe, it is a rule to prepare the patient for decannulation. This part of the treatment is accomplished by corking the tube partially and slowly diminishing the opening of the cork until the tube is totally occluded.

Decannulation in some cases is somewhat troublesome for the following reasons: Fear of choking on the part of the child; lack of re-education of the larynx; insufficient space around the tube.

In this particular patient decannulation by the usual technique was found impossible for this last reason. To overcome this difficulty, an obdurator was devised by Dr. Tucker. This obdurator is of the same size as a No. 1 inner cannula, and it has two distinct advantages. First, permits enough air to go through the larynx; and second, maintains an opening for an emergency, so that a cannula, No. 1, can be slipped into the trachea.

Complete dilatation was accomplished in 78 sittings, taking, altogether, 58 minutes. The child was then able to leave the hospital, with a perfect voice and airway.

CONCLUSIONS.

Stenosis of the larynx can be cured by direct dilatation alone when great destruction of the larynx has not been done, with perfect recovery of all laryngeal functions.

Second, Tucker's tracheal obdurator enables us to overcome the difficulties of decannulation due to insufficient space around the tube.

19th and Lombard Streets.

REPORT OF THREE CASES OF TUBERCULOUS LARYNGITIS TREATED WITH ARTIFICIAL SUNLIGHT.

DR. M. L. HARRIS, Brooklyn.

The object of this presentation is to show the therapeutic value of artificial sunlight in tuberculous laryngitis. The use of the sun's rays for this purpose is not new, neither is that of its substitute, artificial sunlight. It is the ultraviolet rays in the light which have the therapeutic properties. The method in use at the Beth Israel Clinic is that of the carbon arc lamp devised by Dr. Wesseley, which cools the rays by passing them through a water compartment and reflecting them into the larynx by means of a metal laryngeal mirror affixed to the upper jaw by a sort of bridle. Metal laryngeal mirrors are used because they reflect the greatest amount of rays. The wave lengths of the ultraviolet rays produced by this lamp are between 2800 and 3100 Angstrom units. Occasionally, owing to a drooping epiglottis, direct laryngoscopy is resorted to with a Seiffert self-retaining Universal laryngoscope and the rays are applied directly to the lesion; that is, without reflection. This procedure is also more efficient but can only be used where the epiglottis is intact. The patient lies on his back on a table during treatment and the exposure is also shorter.

Tuberculosis of the larynx is practically always secondary to pulmonary phthisis. Infection takes place through the continuous bathing of the laryngeal mucosa with secretion that is coughed up, containing tubercle bacilli. These lesions vary a great deal, from slight redness and thickening of the cords or interarytenoid space to tumefaction of the arytenoids, ventricular bands and epiglottis. Sometimes we get marked deformities and distortions. Frequently ulcerations are produced either on the epiglottis or arytenoid region or cords, which give rise to dysphagia. It is in this phase of tuberculous laryngitis that irradiation with artificial sunlight is most valuable.

The dysphagia prevents proper and sufficient ingestion of food, which detrimentally affects the patient's nutrition and lowers his resistance to the disease. If we can enhance the nutrition of the patient, which is an important part of the treatment of tuberculosis in general, the outcome is much more favorable and it is sometimes

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the decisive factor for recovery. It is well known that the tuberculous patient is hopeful and cheerful until the larynx is involved. He then presents a picture of misery and hopelessness. This is relieved when he can again eat without pain. After treatment the lesions undergo resolution and frequently perfect healing takes place. If the ulcerations can be reached with the ultraviolet rays we can expect epithelialization and healing, although deformities may persist. We rarely use any other therapeutic measure in tuberculous laryngitis.

Dr. Joseph W. Miller, when working in Dr. Wesseley's Clinic in Vienna, was so impressed with the efficiency of his lamp that he had one installed at the Beth Israel Hospital six years ago and the expenditure has justified itself manyfold in the relief it has given our patients, and our experience has proved that his enthusiasm was warranted.

The first patient, E. B., female, age 39 years, gave a history of phthisis of four years' duration on Nov. 1, 1930, when she was first seen. Two weeks before, she became hoarse. Clinically she showed involvement of the upper third of the left lung. The X-ray revealed a fibroid phthisis of the left lung. Biopsy of swollen tissue over right arytenoid was reported tuberculous. The larynx showed marked swelling of the right arytenoid and interarytenoid infiltration. The right cord was red and thickened. Her Fahreus sedimentation test showed a sedimentation time of 6 per cent. This is normal. Her sputum failed to show tubercle bacilli. She has been irradiated from Nov. 6, 1930, to Feb. 24, 1931. Her larynx is much improved and her voice is nearly normal.

The second patient, M. S., age 51 years, a carpenter, came to our clinic, April 9, 1929, complaining of hoarseness for seven months and dysphagia for three months. He gave a history of cough and asthmatic attacks for three years. Tubercl bacilli were found in the sputum. Clinically he showed a bilateral apical lesion, marked emphysema and bronchiectasis. The X-ray showed cavitation at both apices and numerous small foci diffusely scattered throughout the lungs and fibrosis in peripheral portion of the right lung. The larynx showed the epiglottis thickened to twice its normal size and distorted, and ulceration on the laryngeal surface near the free border. The arytenoids were infiltrated to twice the normal size, with ulceration on the left cord posteriorly. The ventricular bands were hypertrophied. Fahreus sedimentation test was 12 per cent.

He was irradiated from April 11, 1929, to January, 1931, about three times a week. About three weeks after treatment was started

his dysphagia was gone. At the end of eight months he had gained 30 pounds. The pulmonary lesions remain the same. The larynx on January, 1931, is so improved that there are no ulcerations, the tissues approximate the normal in size, although there is some deformity of the epiglottis and still some interarytenoid infiltration.

The third case illustrates that clinical improvement can take place with only slight improvement in the lesion in the most malignant type of tuberculous laryngitis. F. L., female, age 30 years, came to the clinic, complaining of hoarseness and dysphagia of six weeks' standing, inability to eat and growing weakness. Chest examination revealed tuberculosis of the lungs and the X-ray findings indicated fibroid phthisis.

The larynx showed marked infiltration of the arytenoids to about three times the normal and the epiglottis swollen to five times its normal size and covering the glottis. The aryepiglottic folds were much swollen and inflamed and there were ulcerations in the pyriform sinuses. Tubercle bacilli were not found in the sputum. She was put on sunlight treatment Oct. 1, 1929, and after about 12 irradiations her pain was gone. May 27, 1930, eight months later, the social service thought it advisable to return her for sanitarium treatment, where the dysphagia returned and she came back to us for treatment on Nov. 18, 1930, after a lapse of six months. Treatment was resumed with alleviation of her dysphagia and some recession of her laryngeal lesion. She is happy and hopeful again.

590 Eastern Parkway.

A CASE OF SEVERE CARDIOSPASM MISTAKEN AND TREATED FOR CARCINOMA OF THE ESOPHAGUS*†

DR. JOSEPH MILLER, New York.

The importance of endoscopy as an aid in diagnosis of esophageal disease is best illustrated by the following case: I. K., an adult male, white, age 49 years, was admitted to the Beth Israel Hospital, service of Dr. Kopetzky on July 23, 1929, with a diagnosis of carcinoma of the esophagus.

About two and one-half years before admission patient had a great deal of psychic trauma, occasioned by business reverses and death of a child. His wife then became mentally deranged.

Following these reverses patient began to complain of a dull pain over sternum at level of fifth rib. He vomited solid food and gradually pain and vomiting became more severe. One year before admission he began to lose weight and soon began to vomit liquids also. He lost 60 pounds in one year. The pain was dull and did not radiate and there was progressive loss of strength. X-rays were taken and a diagnosis of tumor of the esophagus was reported.

With this diagnosis he was taken to a hospital, where a gastrostomy was performed and a tube inserted into the gastronomic fistula. Since no food could pass down the esophagus the patient was fed entirely through the artificial opening of the stomach. He remained in the hospital for three weeks and was then advised to go to the General Memorial Hospital for the express purpose of receiving deep Roentgen therapy.

Thirteen weeks after the operation, the patient, unimproved, applied for admission to the Beth Israel Hospital. The X-ray examination, made on the twenty-fourth and reported on the twenty-fifth, before esophagoscopy, read as follows: There is a complete obstruction at the lower end of the esophagus. There is a dilatation of the esophagus above the obstruction. There is a new growth at the lower end of the esophagus.

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†From the Department of Broncho-Esophagoscopy, Beth Israel Hospital, New York City; service of Dr. Samuel J. Kopetzky.

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On July 25th, two days after admission, I performed an esophagoscopy under local anesthesia. Moderate spasm was encountered at the mouth of the esophagus, which yielded to slight pressure. The successive parts of the esophageal lumen down to the diaphragmatic constriction were found adequate and the mucosa normal. Here definite resistance was encountered, which prevented the advance of the tube further. This obstruction seemed to be a constriction due to spasm rather than a growth. The mucosa in this area was found to be markedly hypertrophied, congested and thrown into folds resembling very much the sphincter ani. There was no sign of ulceration, tumefaction or any other lesion. There was no sign of bleeding.

The X-ray report following the esophagoscopy read as follows: Examination of the esophagus shows a marked dilatation of the esophagus and a point of narrowing at the cardia, the appearance being suggestive of a cardiospasm. There is no new growth of the lower end of the esophagus.

On the day following the esophagoscopy I instituted treatment in the form of retrograde dilatation. I had the patient swallow a braided silk cord, facilitating the passage of the cord with frequent sips of water. When I saw him the following morning he had swallowed 24 inches of the silk. I then proceeded to grasp the distal end of the cord and bring it out through the gastronomic fistula. This was easily accomplished with the Bruening's serrated hook. A bundle of 12 braided silk threads was then tied to the distal end of the silk line and traction made on its proximal end until the bundle was carried well into the constricted area of the esophagus. The other end of the silk lines were kept outside the wound and attached to the abdomen with adhesive plaster and the wound dressed. Instructions were given to feed the patient fluids by mouth and discontinue the feedings through the tube. For the first time in 14 weeks the patient began to take his food by mouth.

On Aug. 1, *i. e.*, five days later, I tied a piece of tape to the silk bundle and pulled the silk and tape out through the mouth, discarding the silk and leaving the tape in the esophagus. On Aug. 8 I tied two strips of tape to the distal end, pulled it upwards into the constricted part of the esophagus. After this semisolid food easily passed down the esophagus into the stomach. On Aug. 16 I removed the tape entirely and allowed the wound to heal. From this time on he consumed solid food and gained weight and strength rapidly.

Gastroscopy was performed on Aug. 23, using a Jackson full-sized gastroscope. Moderate spasm was encountered at the diaphragmatic constriction but the tube soon entered the stomach without difficulty.

He was discharged Aug. 26 and remained well for several months. However, on March 17, 1930, he was readmitted to the hospital, this time with a diagnosis of cardiospasm. On this occasion, his history stated that for the past three weeks he had vomited a great deal of fluids, which came up almost immediately and that he had lost seven pounds in weight. But the symptoms were very mild in comparison with those he suffered during his previous stay in the hospital. Whereas, formerly he had vomited everything he swallowed, this time he managed to retain "about 75 per cent of the solids he ate." Moreover, he had almost complete relief from the pain he suffered before treatment. He still complained of a mild substernal pain, a raw feeling, which vanished in the course of an hour. He had a feeling of the food passing over a raw surface and after it passed he had relief.

Esophagoscopy was performed on March 14 and after a little effort I succeeded in passing the scope through the constricted area into the stomach.

X-ray examination on March 20 revealed a cardiospasm with marked dilatation of the entire esophagus. I then decided to dilate the constriction with Plummer's hydrostatic cardiospasm dilator and accordingly, on March 21, I passed the dilator with the smallest olive attached right through the obstruction and into the stomach. Water was passed into the dilatable part to five pounds pressure and kept there for five minutes.

This procedure was repeated on March 28 and again on April 3. He left the hospital on April 5, 1930, and has had no recurrence of his symptoms since. Last I heard from him was on March 9, 1931, from Los Angeles, Cal., where he is working and in the best of health.

305 East 17th Street.

STAMMERING—A BIBLIOGRAPHY OF THE PAST DECADE—1921 TO 1930, INCLUSIVE.

DR. C. S. BLUEMEL, Denver, Colo.

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Where foreign titles are translated into English, the translations are those given by The Quarterly Cumulative Index Medicus of The American Medical Association.

ANDERSON, L. O.: Preliminary Report of an Experimental Analysis of Causes of Stuttering, *Jour. Applied Psychol.*, v: 340, 1921-22. Stuttering and Allied Disorders, Comparative Psychology Monographs, 1923.

APPelt, A.: Die wirkliche Ursache des Stotterns und seine dauernde Heilung, Münich, 1925.

Stammering and its Permanent Cure, 3rd ed., New York, Dutton, 1930. Treatment of Stuttering, Internat. Ztschr. f. Individual psychol., viii: 125, 1930.

ARISTOFF, V. M.: Success in the Treatment of Stuttering, *Vrach. gaz.*, xxxiv: 549, 1930.

ASH, E. L.: Facts About Stammering, London, Mills and Boon, 1925.

ATKINS, H. A.: Casting out a "Stuttering Devil," *J. Abnorm. Psychol.*, xviii: 137, 1923.

BARNARD, R. H.: Relation of Intelligence and Personality to Speech Disorders, *Elementary Sch. Jour.*, xxx: 604, 1930.

BEHNKE, K. E.: Behnke's Stammering, Cleft Palate Speech, Lisping, 2nd ed. London, Baillière, Tindall and Cox, 1922.

BERGER, W.: Organic and Functional Disorders of Speech, *Ztschr. f. Laryngol., Rhinol.*, xix: 80, 1929.

BLANTON, S.: Disorders of Speech, Minn. Med., ix: 35, 1926.

Medical Significance of the Disorders of Speech, *Jour. A. M. A.* lxxvii: 373, 1921.

Speech Correction Program for the Public Schools, Nat. Conf. Soc. Work, p. 408, 1923.

Speech Defects in School Children, *Ment. Hyg.*, v: 820, 1921; also Nat. Conf. Soc. Work, p. 401, 1921.

Speech Difficulties, *Ment. Hyg.*, xiii: 740, 1929.

Treatment of Stutterers, *Am. Ann. Deaf*, lxvii: 371, 1922.

Working Bibliography for the Beginner in Speech Correction, *Quart. Jour. Speech Education.*, x: 37, 1924.

BLUEMEL, C. S.: Mental Aspects of Stammering, Baltimore, Williams and Wilkins Co., 1930.

Stammering—A Bibliography of the Past Decade—1911 to 1920, Inclusive, *Laryngoscope*, xxxi: 709, 1921.

BORDEN, R. C., and BUSSE, A. C.: Speech Correction, New York, Crofts, 1925.

BREHM, F. E.: Speech Correction, *Am. Ann. Deaf*, lxvii: 361, 1922.

BRILL, A. A.: Speech Disturbances in Nervous and Mental Diseases, *Quart. J. Speech Educ.*, ix: 129, 1923.

BROWN, F. W.: Mental Hygiene of Speech. Correcting and Preventing Speech Disorders, *Ment. Hyg. Bull.*, viii: 1, 4, 1930.

Some Mental Hygiene Aspects of Certain Cases of Stuttering, *Jour. of Expression*, iv: 17, March, 1930.

BURKHARDT, H.: Psychische Ursachen des Stotterns, *Ztschr. f. pädagog. Psychol.*, xxiii: 207, 1922.

BUSSE, A. C.: See Borden, R. C.

- CAMP, P. B.: Speech Correction in Wisconsin Public Schools, *Quart. Jour. Speech Educ.*, ix: 280, 1923.
- CARRIE, W.: Die Bekämpfung des Stotterns durch die Schule, *Ztschr. f. Behandl. Schwachsinn.*, p. 81, 1925.
- Stotternde Schulkinder, *Ztschr. f. Gsndhtsfurs. u. Schulgsndhtspfleg.*, xxxvi: 214, 1923.
- CHADWICK, M.: Stammering, *Canadian Nurse*, xxvi: 65, 1930.
- CHAPIN, A. C.: Organization of Speech Correction Classes in the Los Angeles Public Schools, *Quart. Jour. Speech Educ.*, xiii: 24, 1927.
- CHERVIN: Diagnostic du Bégaiement, *Rev. méd. de l'Est.*, xlvi: 660, 1920.
- CLARK, L. P.: Confirmed Stammerer and His Defective Adaptations in the Nursing Period, *Jour. Nerv. and Ment. Dis.*, lxi: 280, 1925.
- Reeducational Treatment of Confirmed Stammerers, *Med. Rec.*, ci: 609, 1922; also *Arch. Pediat.*, xxxix: 666, 1922.
- Study of Psychogenesis of Confirmed Stammerers, *Jour. Nerv. and Ment. Dis.*, lxiii: 238, 1926.
- COREY, C. B.: Speech Defects in Psychoneurotics, *United States Veterans' Med. Bull.*, i: 10, 1925.
- CORIAT, I.: Active Therapy in Analysis of Stammering, *Psychoanalyt. Rev.*, xvii: 342, 1930.
- Oral-erotic Components of Stammering, *Internat. Jour. Psycho-analysis*, viii: 56, 1927.
- Stammering: a Psychoanalytical Interpretation, New York, Nervous and Mental Disease Publishing Co., 1928.
- CREASY, H. M.: Stuttering, *Arch. Neurol. and Psychiat.*, iv: 713, 1920; also *Jour. Nerv. and Ment. Dis.*, iii: 422, 1920.
- DAHMANN, H.: Speech Defects, *Ztschr. f. Laryng., Rhinol.*, xix: 260, 1930.
- DAITO, T.: Stammering in Children: Familial Speech Disturbance of Guttural Sounds, *Ztschr. f. Hals-, Nasen-u. Ohrenh.*, xxiii: 263, 1929.
- DASHIELL, D. H.: Stammering. Its Cause and Treatment, *Oralism and Auralism*, ii: 29, 1923.
- DELFINO, V.: Orthophonic Therapy of Stammering, *Siglo méd.*, lxxxiv: 402, 1929.
- DEPARREL, G.: Speech Correction and Reeducation, *Presse méd.*, xxxvii: 643, 1929.
- DORATH, J.: Treatment of Stammering by Hypnosis, *Med. Welt.*, ii: 1532, 1928.
- DWORAK, K.: Zwei Fälle von universellum Stammeln und schlechtem Singen, *Eos*, xviii: 104, 1926.
- DYLEWSKI, B.: Disturbances of Speech and Voice in School Children, *Polska gaz.*, ix: 614, 1930.
- FAGAN, L. B.: Certain Reflexes During Stuttering, *Arch. Neurol. and Psychiat.*, xix: 1006, 1928.
- FARMA, W. J.: Speech Disorders and the Teaching of Speech. *Quart. Jour. Speech Educ.*, xii: 156, 1926.
- FERRERI, G.: Phonetic Respiration of Stammerers, *Boll. d. mal. d. orecchio d. gola e d. Naso*, xlvi: 101, 1930.
- FISCHER, A.: Zur Theorie des Stotterns, *Ztschr. f. pädagog. Psychol.*, xxiv: 372, 1923.
- FLATAU, T.: Causes of Stuttering, *Med. Welt.*, iii: 1241, 1929.
- Psychotherapy and General Treatment of Stuttering, *Med. Welt*, iii: 1798, 1929.
- FLETCHER, J. M.: Problem of Stuttering; a Diagnosis and Plan of Treatment, New York, Longmans Green, 1928.
- FOGERTY, E.: Stammering, New York, Dutton, 1930.
- FONTES, V.: Etiology and Clinical Aspects of Stuttering, *Arq. de Anat.*, xiii: 129, 1929.
- Quelques Considerations sur le Bégaiement, *Monde méd.*, xxxx: 831, 1925.
- Sexual Psychology of Stammerers, *Lisboa méd.*, vii: 246, 1930.
- FOSSLER, H. R.: Disturbances in Breathing during Stuttering, *Psychol. Monograph No. 1*, xl: 1, 1930.
- FRANK, D. B.: Intentional Stuttering and Lucidity of Thought Processes, *Ztschr. f. ges. Neurol. u. Psychiat.*, cxxiii: 47, 1930.

- FRANK, L.: Treatment of Stammering by Quacks and Official Medicine, Schweiz. med. Wchnschr., iv: 921, 1925.
- FROMENT, J.: Bégalement et son Mécanisme psychophysiolique, Lyon méd., cxxx: 357, 1921.
- Comment étudier des Troubles du Langage, Ann. méd. psychol., lxxxii: 239, 1924; also Encéphale, xix: 522, 1924; also Rev. neurol., xxxi: 375, 1924.
- Test Words in Dysarthria, Presse méd., xxxii: 1025, 1924 (with P. Ravault).
- FRÖSCHELS, E.: Beitrag zur Symptomatologie des Stotterns, Monatschr. f. Ohrenh., iv: 1109, 1921.
- Curative Pedagogics, Wien. med. Wchnschr., lxxx: 1128, 1930.
- Das Stottern (assoziative Aphasic), München, 1925.
- Diagnosis of Acquired Stammering, Wien. med. Wchnschr., lxxviii: 955, 1928.
- Die herrschenden Ansichten über das Wesen des Stotterns, Arch. f. Psychiat., ixix: 526, 1923.
- Fortschritt der Stotterforschung, Wien. klin. Wchnschr., xxxvii: 82, 1924.
- Nature of Stuttering, Wien. klin. Wchnschr., xlvi: 1096, 1930.
- New Methods of Treatment, Wien. klin. Wchnschr., xli: 1256, 1928.
- Progress in Research of Stammering, Wien. klin. Wchnschr., xxxvii: 82, 1924; abstract, Jour. A. M. A., lxxxii: 756, 1924.
- Stammering, Wien. med. Wchnschr., lxxvii: 1026, 1057, 1927.
- Therapy of Most Important Impediments of Speech, Wien. klin. Wchnschr., xxxviii: 734, 1925.
- Treatment of Speech Defects; Logopedics, Deutsche med. Wchnschr., I: 992, 1924.
- Treatment of Stammering, Klin. Wchnschr., iii: 313, 1924.
- Über die Konstitution assoziativ-aphasischer (stotternder) Kinder, Wien. med. Wchnschr., lxxvi: 873, 1926 (with P. Moses).
- Zur experimentell-phonetischen Diagnostik der Dysarthrien, Ztschr. d.f. ges. Neurol. u. Psychiat., xcii: 487, 1924.
- GIFFORD, M.: Classification and Treatment of Speech Defects at the University of California Hospital, Jour. A. M. A., lxxxii: 1673, 1924.
- Mental and Emotional Reeducation of the Stammerer, Better Health, x: 118, 1929.
- Speech Correction Work in the San Francisco Public Schools, Quart. Jour. Speech Educ., xi: 377, 1925.
- Speech Defects and Disorders, Calif. and West. Med., xxiv: 72, 1926.
- GLASSBURG, J. A.: Importance of Breathing in the Treatment of Speech Disorders, Arch. Otolaryng., ix: 435, 1929.
- Is Stuttering a Medical Problem? Arch. Otolaryng., xi: 430, 1930.
- Is Treatment of Speech Disorders a Medical Problem? Jour. A. M. A., xcii: 958, 1929.
- Stuttering, Am. Med., xxiii: 240, 1928.
- Stuttering; Its Cause and Cure, Arch. Otolaryng., v: 122, 1927.
- Stuttering and Stammering, Med. Times, lvii: 82, 1929.
- What Price Stuttering? Survey, lxi: 363, 1928.
- Why Stutter? Hygeia, vii: 1228, 1929.
- See also Scripture, M. K.
- GOLDBERGER, E.: Treatment of Stammering, Wien. med. Wchnschr., lxxviii: 958, 1928.
- GOLDSTEIN, M. A.: Significance of the Science of Speech Correction, Ann. Otol., Rhin., and Laryngol., xxxv: 161, 1926; also Oralism and Auralism, vi: 14, 1927.
- GÖPFERT, J.: Uebungsbehandlung von Stottern in einem Neurotiker, Lazarett, xxx: 129, 1920.
- GORDAN, M. B.: Stammering Produced by Thyroid Medication, Am. Jour. M. Sc., clxxv: 360, 1928.
- GRAY, G. A.: Straight Talk; Corrective Speech and Better Speech: A Distinction, Educ. R., lxix: 68, 1925.
- GREENE, J. S.: Child's Disordered Speech, Lit. Dig., lxxxiv: 21, Feb. 11, 1925.

- Conclusions Derived from the last Five Years Work at the National Hospital for Speech Disorders, *Bost. M. and S. Jour.*, clxxxix: 57, 1923.
- Departure in Hospitals: the National Hospital for Speech Disorders, *Jour. A. M. A.*, lxxvii: 1726, 1921.
- Problem of the Stutterer, *N. Y. State Jour. Med.*, xxiv: 337, 1924.
- Speech Defects, *Internat. Clin.*, iii: 184, 1930.
- Cause and Cure of Speech Disorders, New York, Macmillan, 1927 (with E. J. Wells).
- GREGORY, M. S.: Stammering as a Neurosis, *Jour. Nerv. and Ment. Dis.*, lxii: 371, 1925.
- GUMPERTZ, F.: Psychology of Stammering, *Med. Klin.*, xxiv: 494, 1928.
- Stammering, *Med. Klinik.*, xxi: 462, 1925; abstract, *Jour. A. M. A.*, lxxxiv: 1608, 1925.
- Ueber den heutigen Stand der Stotter-forschung, *Med. Klin.*, xxii: 264, 1925.
- GUTZMAN, H.: Correction of Phonic Respiration in Stutterers, *Ztschr. f. Laryng., Rhin.*, xviii: 265, 1929.
- Reeducation, particularly in Stammering, *Med. Welt.*, i: 1702, 1927.
- HANSEN, K.: Cure of a Stammering Boy., *Ztschr. f. pädagog. Psychol.*, xxx: 221, 1929.
- HEDRICK, J.: Unique Speech Clinic, *Quart. Jour. Speech Educ.*, viii: 161, 1922.
- HELTMAN, H. J.: Some Educational Aspects of Speech Correction, *Quart. Jour. Speech*, xvi: 221, 1930.
- HERREN, R. Y.: See Travis, L. E.
- IMHOFER, R.: Pathogenesis of Stammering: Neurosis of Expectation, *Med. Klin.*, xxiii: 628, 1927.
- INMAN, W. S.: Inquiry into the Origin of Squint, Left-handedness and Stammer, *Lancet*, ii: 211, 1924.
- JOHNSON, W.: Because I Stutter, New York, Appleton, 1930.
- Child Who Stutters, *Child Welfare*, xxv: 95, 1930.
- Eighteen Years of Stuttering, *Hygeia*, vii: 907, 1930.
- KAIDA, Y.: Psychic Causes of Stuttering, *Ztschr. f. Laryngol., Rhin.*, xx: 48, 1930.
- KAZAKOFF, A. A.: See Toporkoff, N. N.
- KENYON, E. L.: Peripheral Physical Inhibition of Speech, *Arch. Otolaryng.*, xii: 769, 1930.
- KILGOUR, M.: See List, W. E.
- KISTLER, K.: Relation Between Left-handedness and Disturbance of Speech, *Schweiz. med. Wchnschr.*, ix: 32, 1930.
- KITTREDGE, W. B.: Attempt to Discover Another Etiological Factor of Stuttering, *Jour. Educ. Psychol.*, xiv: 162, 1923 (with M. K. Scripture).
- LAMPERT, H.: Medical Care and Educative Methods of Treatment of Stuttering, *Arch. f. Psychiat.*, lxxxiii: 773, 1928.
- LEARY, T. G.: Psychic Aspect of Stammering; its Basis with Corrective Measures, *Med. Jour. Austral.*, i: 141, 1923.
- Stammering and Its Influence on Education, *Med. Jour. Austral.* (Sup.), i: 445, 1924.
- Stammering as a Psychoneurosis, *Med. Jour. Austral.*, i: 299, 1927.
- LEHNER, G.: Differential Diagnosis of Psychogenic Stuttering, *Wien. klin. Wchnschr.*, xxxiv: 476, 1921.
- Stuttering in Relation to Trauma, *Med. Klinik*, xvi: 1059, 1920.
- LEWIS, N. D. C.: Coriat's Stammering, *Psychoanalyt. Rev.*, xv: 443, 1928.
- LIEBMAN, A.: Nature and Treatment of Stammering, *Deutsche med. Wchnschr.*, liii: 800, 1927.
- Reeducation of Stammerers, *Fortsch. d. Zahnheilk.*, iii: 893, 1927; iv: 895, 1928.
- LIMA, M.: Speech Defects in Children, *Ment. Hyg.*, xi: 795, 1927.
- LIST, W. E., and KILGOUR, M.: Speech Clinic at Minneapolis General Hospital, *Jour.-Lancet*, xlii: 331, 1924.
- LOEBELL: Palilalia or Stuttering, *Ztschr. f. Laryng., Rhin.*, xviii: 191, 1929.

- LUKENS, R. M.: Stammering; Clinical Study of 150 Cases, THE LARYNGOSCOPE, xi: 680, 1930 (with M. Trumper).
- MCDOWELL, E. D.: Educational and Emotional Adjustments of Stuttering Children, New York Teachers' College, Columbia University, 1928.
- MANDELL, S.: Psychology of Stuttering, Quart. Jour. Speech, xvi: 200, 1930.
- MARTIN, F.: Curing Stammering in the School, Lit. Dig., lxxi: 62, Oct. 8, 1921.
- Prevention and Correction of Speech Defects, Nat. Ed. Assn., p. 594, 1922; also Eng. Jour., x: 20, 1921.
- MES, L.: Recent Views on Stammering, Geneesk. Gids., vii: 605, 1929.
- MOSES, P.: See Fröschels, E.
- NADOLECZNY, M.: Special Training Schools, Gsndhtsfurs. f.d. Kindesalt., iii: 20, 1928.
- Speech Defects, Klin. Wchnschr., vi: 1409, 1927.
- Stuttering as a Manifestation of a Spastic Coordination Neurosis, Arch. f. Psychiat., lxxxii: 235, 1927.
- NAOR, M.: Phonetic Explanation of Universal Stammering among Children, Ztschr. f. d. ges. Neurol. u. Psychiat., cxxii: 545, 1929.
- NERVEKLUF, T.: Familial Stuttering, Med. Klin., xxiv: 1163, 1928.
- NUSBAUM, E.: See West, R.
- OATES, D. W.: Left-handedness in Relation to Speech Defects, Forum of Ed. (Lond.) No. 7: 191, 1929.
- ORTON, S. T.: Action Currents in Stutterers, Arch. Neurol. and Psychiat., xxi: 68, 1929 (with L. E. Travis).
- Physiological Theory of Reading Disability and Stuttering in Children, New Eng. Jour. Med., cxix: 1046, 1928.
- Stuttering, Arch. Neurol. and Psychiat., xviii: 671, 1927.
- OSWALD, M. V. O.: Stammering and Its Treatment, Med. Press., cxvi (new series) : 172, 1923.
- PACONCELLI-CALZIA, G.: Expressive Gestures in Treatment of Stammering, Ztschr. f. Laryngol., Rhin., xix: 71, 1929.
- PAIKIN, M.: Nature of Stammering, Ztschr. f. Kinderforsch., xxxvii: 508, 1930.
- PALEN, I. B.: Stammering, School., xvii: 631, 1929.
- PALMER, G.: Iowa Conference on Speech Pathology and Experimental Phonetics, Quart. Jour. Speech, xv: 237, 1929.
- PENON, K.: Case of Stammering and Its Treatment, Nederl. Tijdschr. v. Geneesk., lxvi: 722, 1922.
- PEPARD, W. J.: Defects of Speech, THE LARNGOSCOPE, xxxvi: 892, 1926.
- PEPPARD, H. M.: Correction of Speech Defects, New York, Macmillan, 1925.
- Psychology of Stuttering, Sci. Am., cxxvii: 16, 1922.
- QUIDDE, L.: Recollections of a Stutterer, Liv. Age, cccxxviii: 360, 1926.
- QUINAN, C.: Sinistrality in Relation to High Blood Pressure, and Defects of Speech, Arch. Int. Med., xxvii: 255, 1921.
- RAVAULT, P.: See Froment, J.
- RAYMOND, E. M.: What the Government is Doing for Soldiers with Speech Defects, Quart. Jour. Speech Educ., vii: 11, 1921.
- REED, R.: Some Notes on the Stammering Problem, J. Abnorm. Psychol., xvi: 161, 1921.
- RENON, L.: Histoire d'une Cure de Bégalement, Chron. méd., xxviii: 259, 1921.
- ROBBINS, S. D.: Real Cause of Stammering, Med. Rec., xcix: 235, 1921.
- Speech Correction in the Habit Clinic, Bull. Mass Dept. Mental Dis., xiii: 28, 1929.
- Stammering and Its Treatment, Bost. Stammerers Inst., 1926.
- ROMAN-GOLDZIEHER, K.: Handwriting of Stammering Children, Ztschr. f. Kinderforsch., xxxv: 116, 1929.
- Handwriting of Stutterers, Med. Welt., iii: 1244, 1929.
- ROOT, A. R.: Special Education and the Speech Defective, Educ. Adm. and Sup., xiii: 255, 1927.
- Survey of Speech Defectives in the Public Elementary Schools of South Dakota, El. School., p. 531, 1926.

- ROTHE, K. C.: Stammering During Development of Speech a Neurotic Process, *Ztschr. f. pädagog. Psychol.*, xxx: 505, 1929; also *Eos*, xxii: 3, 1930.
- Therapie des Stotterns, *Eos*, xviii: 78, 1926.
- RUMSEY, H. St. J.: Stammering: What is it? *Lancet*, i: 960, 1922.
- No Need to Stammer, New York, Dodd Mead, 1923.
- The Stammering Habit, Guy's Hosp. Gaz., xl: 572, 1926.
- RUSSELL, G. O.: Stammering and Stuttering, Oralism and Auralism, viii: 85, 1929.
- SACHS, M.: Etiology of Stammering, Wien, klin. Wchnschr., xxxvii: 113, 1924; abstract, *Jour. A. M. A.*, lxxxii: 931, 1924.
- Ueben Schielen und Stottern, *Ztschr. f. Augenh.*, liii: 285, 1924.
- SARGENT, P. E.: Schools for Speech Disorders (in his *Handbook of Private Schools*, 1928-29, p. 828).
- SCHICK, A.: Statistical Observations on Stuttering, Wien, klin. Wchnschr., xxxiv: 134, 1921.
- SCHILLER: Speech Defects in School Children, *Ztschr. f. Schulgesundhpflg.*, xlili: 369, 1930.
- SCHOBER, E.: Stammering, *Eos*, xx: 135, 1928.
- SCRIPTURE, E. W.: Das Stottern, *Arch. f. Psychiat.*, lxxii: 108, 1924.
- Elements of Speech Neurology, *Arch. f. Psychiat.*, lxxvii: 573, 1926.
- Experiences at a Speech Clinic, *Practitioner*, cxiii: 381, 1924.
- Nature of Stuttering, *Practitioner*, cxii: 318, 1924.
- Neurological Consideration of Stammering, *Arch. f. Psychiat.*, lxxix: 224, 1926.
- Speech Neurology, *J. Neurol. and Psychopath.*, xi: 156, 1930.
- Stuttering, Lispings and Correction of the Speech of the Deaf (2nd. ed.), New York, Macmillan, 1923.
- Treatment of Stuttering, *Lancet*, i: 749, 1923.
- SCRIPTURE, M. K.: Attempt to Discover another Etiological Factor of Stuttering, *Jour. Educat. Psychol.*, xiv: 163, 1923 (with W. Kittredge).
- Behavioristic Attitudes Caused by Speech Disorders, *Jour. Nerv. and Ment. Dis.*, lxiii: 17, 1926 (with J. A. Glassburg).
- Pathology and Reeducation of Speech Disorders, *Quart. J. Speech Educ.*, xii, 148, 1926.
- Some Theories Concerning Stuttering and Stammering, *Quart. Jour. Speech Educ.*, viii: 145, 1922.
- Stammering and Stuttering from the Medical Viewpoint, *Arch. Pediat.*, xlili: 513, 1925 (with J. A. Glassburg).
- Use of Breathing in the Treatment of Speech Disorders, Oralism and Auralism, v: 59, 1926 (with J. A. Glassburg).
- Use of Relaxation in the Treatment of Speech Disorders, Oralism and Auralism, iv: 124, 1925 (with J. A. Glassburg).
- Use of Vocal Gymnastics in the Treatment of Speech Disorders, Oralism and Auralism, v: 79, 1926.
- SCURI, D.: Etiopathologic Classification of Stammering, *Arch. ital. di Otol.*, xli: 529, 1930.
- SEARL, N.: Stammering in a Child, *Internat. Ztschr. f. Psychoanal.*, xiii: 294, 1927; also *Internat. Jour. of Psycho-Analysis*, viii: 65, 1927.
- SIMEONI, C.: Case of Incoordination between Phonation and Respiration, *Riforma med.*, xli: 796, 1925.
- SOLOMON, M.: How to Treat Stuttering or Stammering, *Hygeia*, iv: 39, 1926.
- Modern Conception of Stuttering or Stammering, *Ill. Med. Jour.*, xlvi: 133, 1925.
- Speech Correction (Editorial), *Quart. Jour. Speech Educ.*, xiii: 218, 1927.
- STAMM, E.: Speech Defects in Old Age, Strasbourg méd., lxxxviii: 477, 1928.
- STARR, H. E.: Hydrogen ion Concentration of the Mixed Saliva...as Applied to the Study of the Metabolic Etiology of Stammering, *Am. Jour. Psychol.*, xxxiii: 394, 1922.

- Psychological Concomitants of High Alveolar Carbon Dioxide; a Psychochemical Study of the Etiology of Stammering, *Psychol. Clinic*, xvii: 1, (March), 1928.
- STEIN, L.: Das universelle Stammeln im Lichte der vergleichenden Sprachwissenschaft, *Ztschr. f.d. ges. Neurol. u. Psychiat.*, xciv: 100, 1925.
- Entwicklungsgeschichtliche Deutung der Entstehung des Silbenwiederholens, *Arch. f. Psychiat.*, ix: 573, 1923.
- Error in Prophylaxis of Stuttering, *Wien. klin. Wchnschr.*, xxxv: 461, 1922.
- Stammering and Asthma (reply to Sternberg's article), *Zentralbl. f. inn. Med.*, xlvi: 233, 1922.
- Stammering with Organic Central (Nervous) Lesion, *Med. Klinik*, xviii: 1315, 1922.
- Ueber das universelle Stammeln, *Eos*, xvii: 19, 1925.
- Ueber die verbreitetsten Theorien des Stotterns, *Wien. med. Wchnschr.*, lxxiv: 1467, 1924.
- Zur Kasuistik des kindlichen Stammelns, *Monatschr. f. Ohrenh.*, lvi: 298, 1922.
- STEINHARDT, L.: Ueber das Stottern, Münich, 1925.
- Causes and Treatment of Stammering, *Med. Welt.*, ii: 1854, 1928.
- STERN, H.: Laryngoscopic Examination in Stuttering, *Wien. med. Wchnschr.*, lxxx: 1357, 1930.
- Zur Pathogenese der Dysarthria spastica, *Ztschr. f. Hals-, Nasen-u. Ohrenh.*, xii: 278, 1925.
- STERNBERG, W.: Stuttering in Relation to Asthma, *Zentralbl. f. inn. Med.*, xlii: 946, 1921; abstract, *Jour. A. M. A.*, lxxviii: 1240, 1922.
- STINCHFIELD, S. M.: Preliminary Classification of Speech Defect Terminology, Oralism and Auralism, v: 71, 1926.
- Relation Between Speech Defects, Musical Disability, Scholastic Attainment and Maladjustment, *Quart. Jour. Speech Educ.*, xiii: 268, 1927.
- Speech Pathology, with Methods in Speech Correction, Boston, Expression Publishing Co., 1928.
- STIVERS, C. G.: Need of Standards of Training for Specialists, General Practitioners, and Teachers of Speech Correction, *Jour. A. M. A.*, lxxix: 533, 1922.
- Systematic Training in the Treatment of Incoordinations, especially of Speech, *Quart. Jour. Speech Educ.*, vii: 52, 1921.
- STOCKERT, F. G.: Zur Aetologie der Mitbewegungen beim Stottern. *Ztschr. f.d. ges. Neurol. u. Psychiat.*, lxxxviii: 459, 1924.
- STRATTON, L. D.: Factor in Etiology of a Sub-breathing Stammerer, *J. Compar. Psychol.*, iv: 325, 1924.
- TARTAR, G.: Case of Stuttering as a Problem of Vocational Readjustment, *Jour. Abnorm. Psychol.*, xxiii: 52, 1928.
- THOMS, P.: Die Stellung Trommers zur Krampftheorie des Stotterns, *Eos*, xviii: 74, 1926.
- TOMPKINS, E.: Stammering and Left-handedness, *Med. Rec.*, c: 941, 1921.
- Summary of Stammering, *West. Med. Times*, xlvi: 39, 1923-24.
- Supplemental Notes on the Stammering Problem, *Jour. Abnorm. Psychol.*, xvii: 132, 1922.
- Two Different Views of Stammering, *Bost. M. and S. Jour.*, clxxxv: 780, 1921.
- TOPORKOFF, N. N.: Prognosis in Nocturnal Enuresis, Stammering and Convulsive Tic, *J. Neuropath. i. Psikhist.*, xx: 627, 1927 (with A. A. Kazakoff).
- TRAVIS, L. E.: Comparative Study of Performance of Stutterers and Normal Speakers in Mirror Tracing, *Psychol. Monog.*, xxxix: 45, 1928.
- Disintegration of Breathing Movements During Stuttering, *Arch. Neurol. and Psychiat.*, xviii: 673, 1927.
- Influence of Group upon Stammerer's Speech in Free Association, *Jour. Abnorm. Psychol.*, xxiii: 45, 1928.
- Muscular Fixation of the Stutterer's Voice Under Emotion, *Science* (new series), lxii: 207, 1925.

- Phono-photographic Study of the Stutterer's Voice and Speech, *Psychol. Monog.*, xxxvi: 109, 1926.
- Photographic Study of the Voice in Stuttering, *Arch. Neurol. and Psychiat.*, xviii: 998, 1927.
- Recurrence of Stuttering following Shift from Normal to Mirror Writing, *Arch. Neurol. and Psychiat.*, xxi: 386, 1929.
- Relation Between Faulty Speech and Lack of Certain Musical Talents, *Psychol. Monog.*, xxxvi: 71, 1926 (with M. C. Davis).
- Simultaneous Antitropic Movements of Hands of Stutterers, *Arch. Neurol. and Psychiat.*, xxii: 487, 1929 (with R. Y. Herren).
- TROMNER, E.: Stammering; a New Conception, *Deutsche Ztschr. f. Nervenheilk.*, cxi: 182, 1929.
- TRUMPER, M.: Hemato-respiratory Study of 101 Consecutive Cases of Stammering, 1929. See also Lukens, R. M.
- TWITMEYER, E. B.: Stammering in Relation to Hemo-respiratory Factors, *Quart. Jour. Speech*, xvi: 278, 1930.
- VAN THAL, J. H.: Education of Children Suffering from Defects of Speech, *Ment. Welfare (Lond.)*, x: 5, 1929.
- International Society for "Logopaedia," *Ment. Welfare (Lond.)*, vii: 42, 1926.
- VON STOCKERT, G. F.: Etiology and Treatment of Stammering, *Deutsche Ztschr. f. Nervenheilk.*, xcix: 284, 1927.
- VORHEES, I. W.: Stammering; a Few Notes from the Observations of a Voice Physician, *Eye, Ear, Nose and Throat Monthly*, i: 121, 1922.
- WARD, I. C.: Defects of Speech. Their Nature and Their Cure, New York, Dutton, 1929.
- WARD, L. A.: Organization of the Work in Speech Correction in a State Department of Public Instruction, *Quart. Jour. Speech Educ.*, xiii: 140, 1927.
- WELLS, E. J.: See Greene, J. S.
- WEST, R.: Motor Test for Dysphemia, *Quart. Jour. Speech*, xv: 469, 1929 (with E. Nusbaum).
- Neurological Test of Movements of Facial and Oral Muscles for Stutterers, *Jour. Neurol. and Psychopath.*, x: 114, 1929.
- WIGGLESWORTH, H. E.: Management of the Stammering Child, *Child (Lond.)*, xiii: 104, 1922-23.
- WILHELM, I.: Zur individualpsychologischen Deutung des Stotterns, Wien. klin. Wchnschr., lxxiv: 1475, 1924.
- WOLFE, W. B.: Stuttering, a State of Mind, *Hygeia*, vi: 560, 1928.
- YOUNG, G. K., and E. H.: Prevention and Treatment of Speech Disorders, *Journal-Lancet*, xlviii: 144, 1928.

International Digest of Current Otolaryngology.

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In the Archives of Surgery for September, 1931, Vol. 23, No. 3, Claiborn and Ferris report two cases of plasma cell tumors of the nasal and nasopharyngeal mucosa and review the twelve cases existent in the literature. The chief symptoms are nasal obstruction, difficulty in deglutition, epistaxis and hoarseness. The size of the tumors is reported as quite variable, the consistency firm and the color grey, red or brown depending on the degree of congestion present. Probably most of the plasma cell tumors are benign neoplasms. The authors stress the importance of histological examination of all nasal tumors even though they appear grossly to be polypi or fibromata.

ROSENBERGER.

Bluemel, of Denver, in the *Jour. A. M. A.*, May 30, 1931, presents a paper on "Stammering as an Impediment of Thought." He feels that stammering is not primarily a speech disorder, but that the disability manifests itself in speech because speech is patterned on thought. Thought disturbance is an inability to have the words clearly in the mind. The stammerer's verbal image momentarily drops out of the stream of consciousness, and this loss of imagery blocks the stammerer's speech. In discussing the therapeutic aspect of speech, the author feels that the first step in speech correction is to learn to stop the physical struggle with which he has formerly made his speech-clock. The stammerer is admonished to give his whole attention to mental speech; he is taught to regard the act of speaking as the process of thinking aloud, and, when speaking, he is urged to listen quietly for his thoughts. The pupil's training is mental drill; the technique of the training has been described by the author in a recent textbook.

BOOK REVIEWS.

Manhattan Hospital Eye, Ear, Nose and Throat Nursing. As practiced at the Manhattan Eye, Ear, Nose and Throat Hospital, New York. 12 mo. of 338 pages, fully illustrated. W. B. Saunders Company, Philadelphia and London. 1931. Price \$2.25.

This little nursing manual has been compiled by Dr. Harmon Smith with the assistance of the directors of the various departments of the Manhattan Eye, Ear, Nose and Throat Hospital. The present is the fifth edition and has been brought up-to-date by the addition of new material and the omission of obsolete data. While the book is written primarily for nurses and, as such, is written in semi- or non-technical language, it still remains a very useful reference book and text for medical students and general practitioners.

The various eye, ear, nose and throat examinations, treatments and operations are described with chief emphasis on the role of the nurse in the procedure. Numerous prescriptions in use at the Manhattan Eye, Ear, Nose and Throat Hospital are presented.

The time necessary to read this brief text is well spent by the specialist, general practitioner, medical student and nurse. M. F.

Die Ohrenkrankheiten Des Kindes. By Dr. med. Max Meyer, Professor für Hals, Nasen und Ohrenheilkunde, an der Universität Wurzburg. With thirty-one illustrations, Berlin: Verlag Von S. Karger, Kalstrasse 59, 1930.

This textbook of 166 pages deals primarily with Otology, but stresses the anatomical, physiological and pathological differences which must be kept in mind in treating diseases of the ear in children. The book makes extremely interesting reading because the author presents his material in a semididactic manner. The work is a compilation of many addresses and papers written by the author.

Dr. Meyer has been able to present basic otologic principles of diagnosis and treatment from two points of view, namely, the otologic and the pediatric, and as such his book forms a useful reference for the problems involved.

The text is freely illustrated with anatomical diagrams, photographs and X-ray pictures; the subject matter is well indexed into twelve sections.

General practitioners, pediatricians and otologists will find Dr. Meyer's book of great help in handling the otologic-pediatric cases. M.F.

Pathologie der Oberen Luft-und Speisewege. By Prof. Dr. Felix Blumenfeld, Wiesbaden, und Prof. Dr. Rudolf Jaffe, Berlin, assisted by Priv. Doz. J. Berberich, Frankfurt a/M; Dr. M. H. Corten, Berlin; Prof. Dr. A. W. Fischer, Frankfurt a/M; Prof. Dr. K. Hellmann, Würzburg; Priv.-Doz. Dr. G. Kelemen, Budapest; Priv.-Doz. Dr. H. Leicher, Frankfurt a/M; Prof. Dr. W. Pfeiffer, Frankfurt a/M; Prof. Dr. H. Schlossberger, Berlin-Dahlem, and Prof. Dr. H. Siegmund, Stuttgart. With 242 illustrations. Leipzig: Verlag von Curt Kabitzsch, 1931. Price: Brosch. Rm. 96.

This is the second volume of a work which was first published in 1929. A review of the first volume appeared in THE LARYNGOSCOPE for December, 1929, p. 836.

It will be noted that the authors have obtained additional assistance in the compilation of this second volume.

The chapters on general inflammations are beautifully worked out, but are too prolific for everyday reference. A short chapter on wound-healing and regeneration makes very interesting reading. The final section of the book, on Tumors, is a concise review of the subject as it applies to our specialty.

The complete book of two volumes is a monument to the authors and will undoubtedly be used for reference purposes by every serious student of the literature. M. F.



DR. HUBERT ARROWSMITH.

DR. HUBERT ARROWSMITH.

Dr. Hubert Arrowsmith, distinguished and veteran otolaryngologist, of Brooklyn, died at his home, September 23, 1931. He was born in Brooklyn, June 5, 1865, received his education in his native city, graduated from the Long Island College Hospital where he received his degree in medicine in 1886.

He is survived by his widow, Mrs. Josephine Howard Arrowsmith, and three daughters. He was married June 30, 1900.

Of the outstanding features of his career mention must be made of his excellent pioneer work in bronchoscopy. Dr. Arrowsmith was the founder of the American Bronchoscopic Society and one of its early presidents. He was Vice-President of the American Laryngological, Rhinological and Otological Society in 1927. He was chairman of the Section of Laryngology of the New York Academy of Medicine and a member of the National Associations in Otolaryngology as follows: Fellow of the American Laryngological Association and member of the American Laryngological, Rhinological and Otological Society. He also held memberships in the American Academy of Ophthalmology and Otolaryngology, American Bronchoscopic Society, American College of Surgeons and the American Medical Association.

About two years before his death Dr. Arrowsmith virtually retired from active practice. Prior to that time he was laryngologist to Kings' County Hospital, St. Peter's Hospital, the Brooklyn State Hospital, the Jewish Hospital, the Brooklyn Hospital, and St. John's Hospital, all of Brooklyn. He was also consulting laryngologist at the Huntington Hospital, Huntington, L. I., and St. Anthony's Hospital in Wood Haven, Queens.

We knew him and loved him as a friend and colleague. His was a genial, wholesouled and fairminded nature. He was loyal to his friends, generous and kindly disposed to the younger men in the profession, a severe but just critic, an untiring worker, seriously and constantly interested in the specialties to which he devoted his life. His scientific contributions were mainly in the field of laryngology and bronchoscopy. His discussions at the National Association meet-

ings were few but effective. He was as modest and retiring scientifically as he was in his personal associations, but was always listened to with respect and eagerness.

Laryngology has lost a valiant champion; and we, his colleagues, an esteemed friend.

BIBLIOGRAPHY OF THE LATE HUBERT ARROWSMITH.

1. A Clinical Study of Seven Hundred and One Cases of Nasopharyngeal Adenoids Observed in Two Thousand Dispensary Cases. *N. Y. Med. Jour.*, August 28, 1897.
2. The Tuberculosis Question. *The Med. News*, Oct. 12, 1901.
3. Some Points of Equal Importance to the Dentist and the Rhinologist. *Items of Interest*, Nov., 1908.
4. Vincent's Angina Involving the Larynx Exclusively. *Ann. Otology*, Sept., 1910.
5. A Case of Apparently Primary Intralaryngeal Actinomycosis. *THE LARYNGOSCOPE*, Oct., 1910.
6. Certain Aspects of Rhinolaryngology and their Relation to General Medicine. *N. Y. Med. Jour.*, Dec. 17, 1910.
7. A Case of True Papilloma of the Nasal Septum. *THE LARYNGOSCOPE*, Feb., 1911.
8. Unusual Laryngeal Tumor Specimen. A Curious Esophageal Experience. *THE LARYNGOSCOPE*, May, 1914.
9. A Case of Recurrent Nasopharyngeal Fibroma. *THE LARYNGOSCOPE*, July, 1914.
10. Cavernous Angioma of the Tongue. *THE LARYNGOSCOPE*, Feb., 1915.
11. Angioneurotic Edema of the Esophagus. *THE LARYNGOSCOPE*, Mar., 1915.
12. A Case of Tubercular Leprosy involving the Upper Air Passages. *THE LARYNGOSCOPE*, Mar., 1916.
13. Malignant Hypernephroma of the Ethmoidal Region. *THE LARYNGOSCOPE*, May, 1916.
14. Lye Stricture of the Esophagus. Blind Bouginage, Perforation, Death. *N. Y. Med. Jour.*, June 3, 1916.
15. Tracheobronchial Syphilis. *N. Y. Med. Jour.*, June 24, 1916.
16. Diffuse Diabetic Ulceration of Pharynx and Larynx. *THE LARYNGOSCOPE*, Sept., 1916.
17. New Safety Pin Closer for Endoscopic Use. *THE LARYNGOSCOPE*, Mar., 1917.
18. Peroral Endoscopy in Diagnosis and Treatment, etc., *Trans. Sect. Laryng. Otolog. and Rhinol.*, A. M. A., 1917.
19. Present Day Aspects of Laryngeal Tuberculosis. *N. Y. Med. Jour.*, Aug. 1, 1917.
20. Stone Removed from Esophagus of Insane Patient. *THE LARYNGOSCOPE*, Dec., 1917.
21. The Surgery of Laryngeal Malignancy. *N. Y. Med. Jour.*, June 15, 1918.
22. Laryngectomy for Epithelioma. *THE LARYNGOSCOPE*, June, 1918.
23. The Treatment of New Growths of the Larynx by Internal Surgical Methods. *N. Y. State Jour. Med.*, Sept., 1918.
24. Foreign Bodies in the Air and Upper Food Passages in Pre-Endoscopic Days. *THE LARYNGOSCOPE*, Nov., 1919.
25. Diverticulum of the Esophagus. *N. Y. Med. Jour.*, Jan. 3, 1920.
26. Some Endoscopic Mishaps. *N. Y. Med. Jour.*, June 19, 1920.
27. Traction Pulsion Diverticulum of the Esophagus. *N. Y. Med. Jour.*, Mar. 16, 1921.
28. Gangosa. *THE LARYNGOSCOPE*, Nov., 1921.

M. A. G.

DR. EMIL MAYER.

Dr. Emil Mayer, for more than half a century a distinguished figure in laryngology, died at his home, in New York, October 21, 1931. He was born in New York, May 23, 1854; a student at the College of the City of New York, in 1868 and 1870, he was graduated from the College of Pharmacy in 1873. In 1877 he received his M. D. Degree from the New York University Medical College. He lectured in laryngology at the New York Polyclinic Institute and from 1893 to 1904 was chief of the Throat Department Clinic at the New York Eye and Ear Infirmary. From 1904 to 1919 he was attending laryngologist at Mt. Sinai Hospital and Chief of the Clinic of Nose and Throat department of Mt. Sinai Dispensary.

During the World War, Dr. Mayer was a member of the Medical Advisory Board of the United States Army and Chief of the Medical Intelligence Bureau of the American Red Cross.

He was a Fellow of the American Laryngological Association and its president in 1922; an Honorary Fellow of the American Academy of Ophthalmology and Otolaryngology and its president in 1920, and a former president of the Section of Laryngology and Otology of the American Medical Association.

Dr. Mayer was also chairman of the research committee on local anesthesia, under the Council of Pharmacy and Chemistry of the American Medical Association; a member of the New York Academy of Medicine, the New York County Medical Society and the New York State Medical Association. He was an honorary member of the Philadelphia Laryngological Association, the American Laryngological, Rhinological and Otological Society, the American Stomatological Society, and the American Bronchoscopic Society.

For sixteen years Dr. Mayer was American Editor of the *Zentralblatt für Laryngologie*. He was associated with Dr. James J. King in the writing and editing of a textbook on local anesthesia in head surgery. He contributed more than eighty articles to various medical journals.

He was a frequent contributor to our literature, mainly in the field of laryngology.

His was a delightful personality, accentuated by a fine cultural background. He was a patron of the arts, a collector of etchings, an indefatigable reader, and extremely interested in all phases in the development of the specialty of laryngology; and he served this specialty long, faithfully and well.

M. A. G.

